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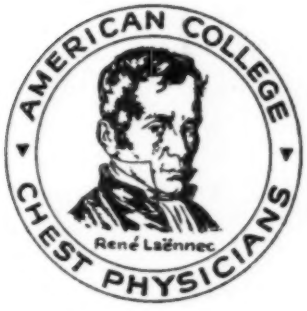

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DISEASES *of the* CHEST

VOLUME XIV JANUARY-FEBRUARY, 1948 NUMBER 1

Tuberculosis of Trachea and Major Bronchi*

HENRY C. SWEANY, M.D.** and HUGO BEHM, M.D.***
Chicago, Illinois

Although tuberculosis of the trachea and bronchi has been known for many years as a complication of pulmonary tuberculosis, its importance with regard to the symptoms, diagnosis, prognosis and treatment of the pulmonary lesion has only been realized within the last decade. Perhaps no one factor has been so important in bringing about this change as bronchoscopy. With the increase in the use of the bronchoscope, many hitherto unsuspected cases of tracheobronchial tuberculosis have been discovered in patients previously thought to have only pulmonary tuberculosis. Notwithstanding the importance of bronchoscopy, it has been necessary to resort to post mortem examination to determine the large incidence of these complications.

We know from clinical reports, based principally on bronchoscopic examinations, that tuberculous tracheobronchitis can change the course of pulmonary tuberculosis from slight to grave and many times fatal symptoms. (Bugher, Littig and Culp,[1] Hawkins,[2] Myerson,[3,4] Samson,[5,6] Barnwell, Littig and Culp,[7] Cohen and Wessler,[8] Jenks,[9] Salkin, Cadden and Edson,[10] Dighiero,[11] and many others.)

Tracheobronchial disease explains the symptoms of clinical obstruction (wheezing, ronchi, asthmatoid attacks, capricious elevations of temperature, changing amount of expectoration, cyanosis, and sudden atelectasis. Jackson,[12] has illustrated the mechanism of stenosis by beautiful color plates. Also this complication accounts

*From the City of Chicago Municipal Tuberculosis Sanitarium Laboratories. Read in abstract form at the VII Pan American Congress of Tuberculosis at Lima, Peru, March 17th, 1947.

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for the persistent positive sputum and the sudden bronchogenic spread which is observed sometimes in cases apparently arrested or in cases receiving pneumothorax. Probably the most important symptoms of bronchial lesion are related to bronchial stenosis and, consequently, to the disturbed passage of air in the bronchus (Tuttle, O'Brien, Day and Phillips,[13] Eloesser,[14] Allison,[15] Brantigan, Hoffman and Proctor,[16] etc.). In fact, stenosis seems to be the physio-pathological reason for the emphysema and atelectasis, which have a variable evolution and distribution and which hold an important place in the diagnosis. Bronchial stenosis is particularly important in the evolution of the pulmonary cavities and also determines whether or not cavity closure or cavity distension follows, as has been shown by Coryllos,[17] Pagel and Simmonds,[18] Shipman[19] and others.

The development of tracheobronchial tuberculosis is a serious complication which always causes a worse prognosis, especially if stenosis results. Available statistics gathered by Tuttle and associates[13] show that 32.6 per cent of the recorded cases of bronchial stenosis proved fatal. It was reasonable to asume, therefore, that only two-thirds of the ulcero-stenotic bronchial tuberculosis has a chance to improve.

Likewise, treatment of the disease has been made more difficult and hazardous because of tuberculous tracheobronchitis. Rest treatment alone does not often relieve the condition and collapse therapy is sometimes dangerous. Authors are not wholly in agreement concerning the use of collapse therapy, especially the particular form of collapse. The concensus of opinion is that pneumothorax is detrimental and that thoracoplasty generally gives good results. (Alexander, Sommer, Trenton and Ehler,[20] Tuttle, et al,[13], Samson,[5,6] Chamberlain and Gordon,[21] Rafferty and Shields,[22] Eloesser,[14] however, was not so sure that thoracoplasty is of great benefit.

Among the numerous reports on this subject, McIndoe, Steele, Sampson, Anderson and Leslie[23] were perhaps the first to make a systematic bronchoscopic examination of non-selected cases of pulmonary tuberculosis. In 272 routine admissions they found 11 per cent of tracheobronchial tuberculosis—a figure that has been approached by nearly all subsequent reports on unselected cases of tuberculosis. (Salkin, Cadden and Edson,[10] McCullough,[24] Wilson,[25] and others). Sharp and Gorham[26] in 53 cases studied in the same way, however, described 37 per cent of positive findings. Acuña[27] reported 29.7 per cent of the complication in 279 bronchoscopic examinations. Other reports also give higher figures, but it is probable that most figures over 15 per cent include cases selected because of some suspicious clinical signs.

Regarding the pathological aspect of the subject, Louis[28] was one of the first to describe the pathology of the condition. He reported 20.3 per cent tuberculous involvement of the trachea—a figure not greatly different from recent reports. Eppinger[29] in 1880 reported 8 per cent tracheobronchitis in cases free from laryngitis. Since about two-thirds of cases have laryngitis, the actual incidence in his cases would be around 25 per cent. Cornet[30] in 1904 described a pathological classification. The first extensive study devoted completely to tuberculosis of the bronchial tree was conducted by Habershon[31] in 1905, who reported that in 1255 autopsies, 10.4 per cent had tracheal tuberculosis.

Minkovsky[32] reported in 1929 a study of 2379 autopsies and found 11.48 per cent of tracheal tuberculosis in the gross examination. He distinguishes two types, a productive and an exudative, and admits the possibility of infection of the trachea by different ways (hematogenous, lymphatic and by implantation).

Reichke and Frost,[33] in a small series, attempted to explain a new pathogenetic theory. They observed that many of the early lesions were located in the mucous glands which connect the adventitia with the mucosa. As a result, they postulated that the infection passes to the mucosa from the peribronchial lymphatics through the lymph nodes. They noticed in some cases that the drainage bronchus of tuberculous cavities has no tuberculous lesions even in places very close to the cavity. The authors contend that pathogenesis by implantation is thereby disproved. Ornstein and Epstein[34] reported eight cases of bronchial tuberculosis with no important tuberculous lung lesion. The latter authors feel that the finding supports the Reichle and Frost theory.

Bugher, Littig and Culp[1] studied both microscopically and macroscopically the respiratory tree from the larynx to the major bronchi in 122 cases of pulmonary tuberculosis, 25 of which were miliary. In 20.5 per cent tracheal tuberculosis, 33.6 per cent bronchial, and 30.4 per cent laryngeal, tuberculosis was found. In 76 per cent of the tracheobronchial cases there was also laryngeal tuberculosis, but they claim that the laryngeal lesion is not secondary to the tracheal lesion. Tracheobronchial tuberculosis alone was present in 41.1 per cent of the cases. Since there were 25 cases of miliary tuberculosis, 21 of which were negative for tracheobronchial lesion, it would raise the figure for tuberculous tracheobronchitis in non-miliary cases to almost 50 per cent. Microscopically they described the early lesions as sub-mucous tubercles related to the glands or their ducts. Caseation of these tubercles leads to ulceration. They think that infection results from implantation in the larynx, trachea and main bronchi.

Auerbach[35] gave an excellent description of bronchial tubercu-

losis but reported only 4.4 per cent positive findings. He described isolated miliary tuberculosis, ulcerative forms and caseous bronchitis. Flance and Wheeler[36] studied the respiratory tree from the trachea to the third order of bronchi in 285 cases of pulmonary tuberculosis and found only 3.1 per cent of local lesions.

Among the more recent works on the pathology of tracheobronchial tuberculosis, Perez[37] studied grossly and microscopically most of the respiratory tree in ten cases of far advanced pulmonary tuberculosis. In nine of them he found a tuberculosis lesion somewhere in the trachea and the bronchi. Heafs[38] reported 44 per cent of tracheal tuberculosis in 133 autopsies.

Wilbur,[39] in an exhaustive study, described 35.4 per cent of tuberculous tracheobronchitis in 500 autopsies, usually located in the postero-lateral wall of the lower portion of the trachea and the adjacent portions of the major bronchi. The author described the early lesions related with the glands as productive tubercles which may develop caseation and form granulatory ulcers. She reasons with admirable logic that the pathogenic mechanism is most frequent by implantation, with the exception of the perforation of lymph nodes in the trachea or bronchi and of the cases of tuberculous tracheobronchitis in the middle of the caseous foci. Meissner[40] reported 52 per cent positive tuberculous tracheobronchitis in 60 specimens of lungs removed by lobectomy. Chia-ssu

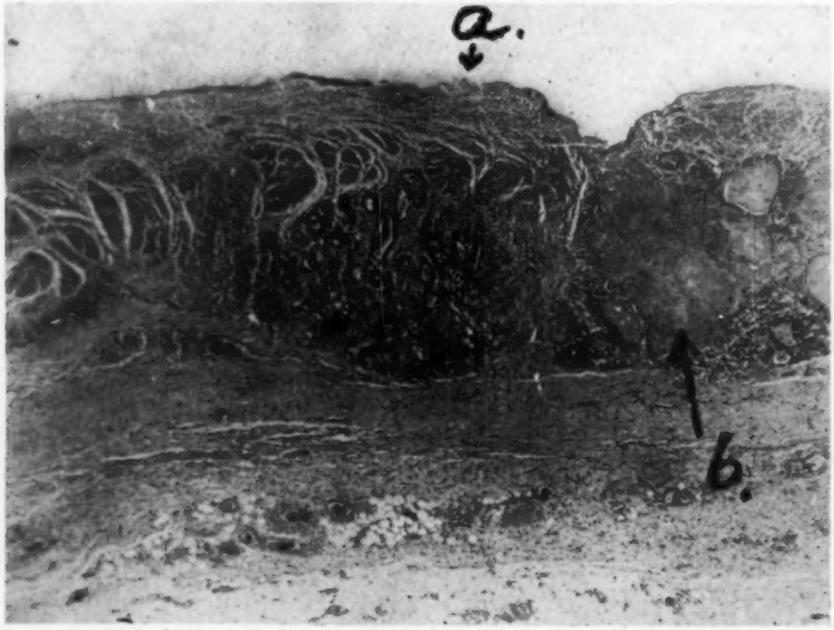

FIGURE 1
A low power microphotograph of the wall of a bronchus showing a superficial ulcer at "a" and gland tubercle at "b". H & E x 20.

Huang[41] reported 42.7 per cent tuberculous tracheobronchitis and 50.4 per cent if laryngitis were included on 115 autopsies.

Material

Our report involves the analysis of 667 autopsy specimens of pulmonary tuberculosis cases in which the trachea and the main bronchi were systematically studied.

General Incidence

As a result, 377 (56.5 per cent) cases of tuberculous tracheobronchitis were found on gross examination. About half of these positive cases (185 specimens) and a fair sample of the negative cases of the trachea and bronchi, were examined microscopically.

In the cases studied microscopically, four sections were taken, one each from the upper and lower trachea, and one each from each main bronchus. The sections were generally taken through regions showing pathological changes. It must be stated, however, that the negative cases were not as thoroughly studied by microscopical section as were the positive cases. Of the 290 negative cases only 32 were examined microscopically, 12 (37.5 per cent) of which were found to be tuberculous. It is clear, therefore, that microscopic search will increase considerably the true tuberculosis incidence, notwithstanding the fact that many of the cases are of little or no consequence. As a result, an estimated figure of tracheobronchial tuberculosis was increased to 486 cases, or 72.9 per cent of the total. Should complete serial sections of the trachea and bronchi be made, even higher figures would possibly be found. Our statistics, therefore, tend to agree with Perez'[37] figures which indicate that in far advanced pulmonary tuberculosis most cases have tuberculosis somewhere in the tracheobronchial tree. In spite of the fact that many of these minimal lesions have no clinical significance, they do prove the extraordinarily widespread of the tuberculous infection in the passages leading out from the lungs.

GENERAL INCIDENCE OF GROSS LESIONS

TABLE I

Sex Incidence

	Males	Females	TOTAL
Positive tuberculous tracheobronchitis	170 (47.4%)	207 (67.9%)	377
Negative tuberculous tracheobronchitis	189 (52.6%)	101 (32.1%)	290
TOTAL	359	308	667

Sex Incidence (Table I)

Females were found to have a higher incidence of tracheobronchial tuberculosis than males, 67.9 per cent of which were positive, while only 47.4 per cent were positive in males. The difference in sex incidence may be related, partly at least, to the higher morbidity and mortality of young females.

Age Incidence (Table II)

The study by age groups demonstrates that the incidence of tracheobronchial tuberculosis decreases progressively with age in such a way that the incidence over 60 years is almost half that of the 11-20 age group (31.25 per cent and 61.05 per cent, respectively). The greatest decrease, however, occurs rather abruptly after 40 years. The higher incidence of these lesions on younger groups might be explained by the more exudative character of

FIGURE 2

A moderately high power microphotograph of a sub-mucous gland duct showing early caseous tubercles and giant cells. Most deep ulcers probably originate in this manner. H & E x 50.

the disease and a probable tendency of the pulmonary tuberculosis to become more generalized in the younger age groups.

Incidence in the Various Stages of the Disease (Table III)

The proportion of positive cases in far advanced, moderately advanced and minimal pulmonary tuberculosis in the series studied are not statistically significant, because there were so few cases in the latter two groups. It is more important to emphasize that 87.85 per cent of the cases studied were far advanced disease and that the extent of parenchymal disease logically might be expected to parallel the extent of involvement in the trachea and bronchi.

The Relation of Tuberculous Tracheobronchitis to Cavitation (Table IV)

The incidence of tracheobronchial tuberculosis as related to the existence of tuberculous cavities in the lung, indicates no important difference between the cavitary and non-cavitary cases. Here

TABLE II

Age Incidence

	0-10 Years	11-20 Years	21-30 Years	31-40 Years
Positive Cases	5 (62.5%)	58 (61.1%)	150 (63.8%)	90 (58.4%)
Negative Cases	3 (37.5%)	37 (38.9%)	85 (36.2%)	64 (41.6%)

(Age Incidence, continued)

	41-50 Years	51-60 Years	60 or more	TOTAL
Positive Cases	48 (45.7%)	18 (33.3%)	5 (31.3%)	374
Negative Cases	57 (54.3%)	36 (66.7%)	11 (68.7%)	293

TABLE III

Relation of the Stage of the Disease to Tracheobronchial Tuberculosis

Stage of Disease	Far Advanced	Moderately Advanced	Minimal	TOTAL
Positive tuberculous tracheobronchitis	340 (58.0%)	35 (50.6%)	6 (50%)	381
Negative tuberculous tracheobronchitis	246 (42.0%)	34 (49.4%)	6 (50%)	286
TOTAL	586	69	12	667
Per cent of all cases	87.5%	10.7%	1.8%	667

again the group of cases with no cavitation is too small because over 90 per cent of all cases have one or more cavities. Finally, the existence of bilateral or unilateral cavities and the fact that the cavities are located in the right or left lung does not seem to have any statistical importance in the origin of the tracheal localization. On the other hand, in the small group of cases having unilateral cavernous pulmonary tuberculosis and unilateral bronchial tuberculosis, it is quite evident that in 85 per cent of the cases the bronchial lesion is homolateral to the lung lesion, showing a close relationship between the two processes.

Location of the Lesions Along the Bronchial Tree (Table V)

There was a diffuse spread throughout the trachea and main bronchi in 64.7 per cent of the cases. Most of the localized lesions were found in the lower segment of the trachea and in the adjoining portion of the main bronchi. When the bronchial lesion was unilateral, usually the affected bronchus was on the same side as the more advanced pulmonary tuberculosis.

TABLE IV
Relation of Number and Location of Cavities to Tuberculous Tracheobronchitis

	No Cavitation	Total Cavernous Cases	Bilateral Cavities	Right Cavities	Left Cavities
Positive tuberculous tracheobronchitis	26 (55.3%)	351 (56.6%)	234 (59.2%)	55 (57.3%)	62 (55.4%)
Negative tuberculous tracheobronchitis	21 (44.7%)	269 (43.4%)	178 (40.8%)	41 (42.7%)	50 (44.6%)

TABLE V
Distribution of Gross Lesions

Generalized distribution		242 (64.7%)
Lesions in lower portions of the trachea and adjacent portions of the main bronchi		65 (17.3%)
Trachea and left bronchus		19 (5.0%)
Trachea and right bronchus		9 (2.4%)
Trachea alone		31 (8.2%)
throughout trachea	17	
upper trachea	6	
lower trachea	8	
Bronchi alone		11 (2.9%)
left bronchus	10	
right bronchus	0	
both bronchi	1	

In Wilber's[39] report a table is presented revealing a wide divergence of opinion regarding the anterio-lateral position of the lesions with many favoring the posterior and posterior-lateral walls as points of predilection for the location of the lesions. Although in our series more ulceration was found in the posterior than anterior half of tree, the difference was not strikingly great. There were many isolated anterio-lateral ulcers. Lesions located in the bronchi alone are much less frequent (2.9 per cent) and the tracheal involvement alone was only present in 8.2 per cent of the positive cases. But, if localized, they may be found as frequently in the upper half as in the lower. The other combinations are presented in the tables.

Degree of Involvement (Table VI)

In an attempt to classify the various lesions according to the extent of involvement, we divided the lesions into "far" and "moderately advanced," and "minimal." The far advanced cases were those in which there was a wide spread extension of the ulceration through the mucosa (many through the adventitial layer, with some perforations). Minimal cases with those having few and small superficial ulcers. The moderately advanced cases were all the rest. According to this classification there were 37.4 per cent far advanced, 44.6 per cent moderately advanced, with only 18.0 per cent minimal. These figures do not include the grossly negative cases where a microscopic study on a small sample revealed tuberculosis pathology in 37.5 per cent of the samples studied. In the gross lesions alone, 72 per cent were rather well or far advanced.

Character of Involvement

In only a few cases did the tracheobronchial tuberculosis consist of an infiltrative lesion without ulceration where a thickened, granulous mucosa was found. Most of tuberculosis of the trachea and bronchi consisted of ulcerations which were variable in nature and extent. The most common ulceration observed was 1-2 mm. in size, with irregular and thin edges, oval or irregular in shape. The base consisted of a granular surface, sometimes covered by a

TABLE VI

The Extent of Involvement of Gross Tracheobronchial Tuberculosis

"Far advanced"	141	(37.4%)
"Moderately advanced"	168	(44.6%)
"Minimal"	68	(18.4%)
TOTAL	377	

grayish exudation. There was a wide variety of ulcerations, however, ranging from the superficial miliary ulcers to giant confluent ulcers with deep erosions of a caseous nature or deep erosions with grayish red granulomatous floor and overhanging edges. On the other hand, there are ulcers which have a more fibrous appearance. In a few cases there were fibrous scars which indicate a healed ulcer. The majority of the lesions are confined to the mucous layer, with a less number to the submucosa, and with only a small percentage extending to the adventitious layer.

Caseous Bronchitis (Table VII)

Four cases had the type of a caseous bronchitis, wherein extensive caseous lesions had extended throughout the mucosa and submucosa along large segments of the bronchus.

Fibrous Stenosis

Excluding the cases in which the inflammatory lesion had produced a narrowing of the bronchi, we observed five typical cases of fibrous stenosis, most of them with ulcerative lesions in the

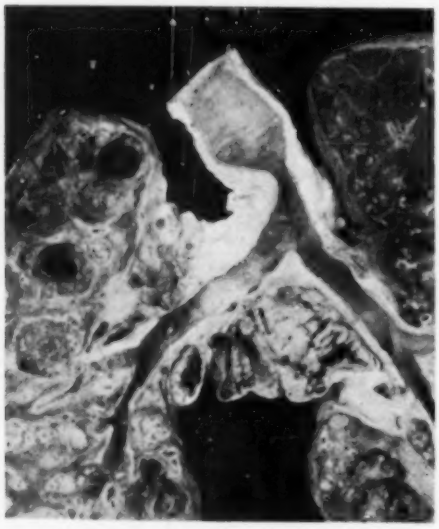

FIGURE 3: A primary tuberculous tracheo-bronchitis of the left main bronchus and the lower part of the trachea. The acute phase has passed, but the effects of obstruction may be seen in the bronchiectatic cavities out in the lung and the stricture of the trachea.

TABLE VII

Coexistence of Tracheo-Bronchial Tuberculosis and Tuberculosis of the Larynx in 621 Cases Having Laryngeal Examinations

Gross tuberculous tracheobronchitis	Laryngeal tuberculosis	No gross tuberculous tracheobronchitis	Laryngeal tuberculosis
355	210 (59.1%)	266	106 (39.8%)

Of 316 cases of tuberculous laryngitis 210 (66.5%) had tuberculous tracheobronchitis.

mucosa. In four of these cases the stenosis was located in one bronchus; the other case was a stenosis involving the middle third of the trachea. The degree of stenosis ranged from 20 per cent to 70 per cent of the lumen. Only in one case was found atelectasis in the corresponding lung. We have not considered in this group the bronchial tuberculosis located in a narrowed bronchus because of surgical collapse or a pleural effusion.

Lymph Node Perforations

Finally, six cases were found to be lymph node perforation in the trachea or main bronchi. Two were located in the left main bronchus, two in the right major bronchus, one in the upper portion of the trachea, and one in the lower. In all of them there was a caseous tuberculosis of the respective lymph node.

Tracheobronchial Tuberculosis and Laryngeal Tuberculosis (Table VIII)

Of the 667 cases, only 621 had a laryngeal examination. In 316 cases with laryngeal tuberculosis a simultaneous tracheo-bronchial

TABLE VIII

Gross Character of Lesions

Diffuse superficial infiltration	12 (3.2%)
Diffuse deep infiltration with ulceration	85 (22.8%)
Circumscribed ulceration	265 (70.3%)
Stenosis (healed ulceration)	5 (1.3%)
Perforation (lymph node)	6 (1.6%)
Caseous bronchitis	4 (1.0%)
TOTAL	377 (100%)

TABLE IX

Microscopic Character of Selected Cases Having Positive Gross Lessions

With submucous epithelioid tubercles and giant cells only	38 (20.5%)
With specific ulceration	97 (52.4%)
Specific ulceration with fibroid cavernous walls	20 (10.8%)
Non-specific ulceration	15 (8.1%)
Non-specific ulceration with fibroid cavernous walls	2 (1.1%)
Fibrotic lesions with isolated specific foci	4 (2.2%)
Extensive caseation	4 (2.2%)
Non-ulcerative non-specific infiltration	5 (2.7%)
TOTAL	185 (100%)

tuberculosis was found in 210 cases or 66.45 per cent. On the other hand, in 355 cases with tracheobronchial tuberculosis there were 210 or 59.1 per cent with tuberculosis of the larynx. In 266 cases with no tracheobronchial lesions only 106 cases (39.8 per cent) had ulcerations. In far advanced cases the two lesions usually were simultaneous in the larynx and the tracheobronchial tree.

Microscopic Character of the Lesions (Table IX)

The sample including nearly 50 per cent of all gross lesions, was classified according to the microscopic findings. There were 38 cases (20.5 per cent) having only submucous epithelioid tubercles and giant cells with no evident break in the mucosa. Acute specific ulceration was present in 97 cases (52.4 per cent). Chronic specific ulceration was present in 20 cases (10.8 per cent). Non-specific ulceration in which no epithelioid tubercles, giant cell or typical caseation were found, was present in 15 cases (8.1 per cent). Other findings included non-specific ulceration with fibroid walls, 2 cases (1.1 per cent); fibrotic lesions with isolated specific foci, 4 cases (2.2 per cent); extensive caseation, 4 cases (2.2 per cent), and non-ulcerative non-specific infiltration, 5 cases (2.7 per cent).

FIGURE 4

(a) Showing a diffuse tracheo-bronchitis and laryngitis. Discrete ulcers cannot be seen but there is a continuous caseous process throughout.

(b) A healed tracheitis showing strands of fibrous tissue crossing the cartilages.

Control Cases

We have already mentioned the control group of 32 negative cases where 12 (37.5 per cent) microscopically tuberculous lesions were found. Usually the lesion consisted of epithelioid tubercles located in the submucosa or miliary caseated foci with early ulceration and specific granulation.

In another group with tuberculous lesions apparently localized to a portion of the tracheobronchial tree, sections were saved at different levels where the mucosa seemed to be normal. In 29 of this type of cases, 12 or 34.5 per cent were found to have microscopic tuberculous lesions. As we have already pointed out, these facts show how widely disseminated are the lesions when the pulmonary tuberculosis is far advanced.

DISCUSSION

There are several features concerning tuberculous tracheobronchitis in general and this study in particular that needs clarification.

One of the most perplexing problems is the disparity of figures given in the literature. For example, it is difficult to reconcile the findings of Bruhig, Littig and Culp[1] who report nearly 50 per cent positive findings, with those of Flance and Wheeler who report only 3.1 per cent; or Wilber's 35 per cent with Auerbach's 4.4 per cent. Figures generally range between these extremes. A few reports, however, may be corrected to give a more uniform result.

For example, Eppinger found only 8 per cent positive tuberculous tracheobronchitis on autopsy material, but he did not include associated tuberculous laryngitis. If a correction is made for the association of the two conditions, the result would be around 25 per cent for all tuberculous tracheobronchitis because more than two-thirds of the cases of tracheobronchitis also have laryngitis.

After weighing all the evidence it is becoming increasingly clear that few cases of far advanced pulmonary tuberculosis are entirely free from tracheobronchial lesions especially after a rigid microscopic search. Since most pulmonary cases become advanced before death, it follows that the majority of cases coming to autopsy should reveal some tuberculous tracheobronchitis. We feel, therefore, that any reports over 50 per cent positive are within the range of the true incidence. The variations above that figure depend upon the percentage of far advanced cases and the care used by workers in finding all the lesions.

Bronchoscopic studies on clinical material also reveal a wide variation in results but the differences can generally be harmon-

ized by considering the type of material. For example, McIndoe and associates[23] report 11 per cent on the examination of *routine* admissions while Sharp and Gorham[25] report 33 per cent on cases selected largely as a result of some distressing symptom or symptoms. Differences would also be present due to the difference in stages of the disease admitted to various institutions.

It seems logical that the young ages should be more prone to be affected than are older age groups. In fact, the general tendency to produce exudative lesions, as in the teen ages, female sex, and aboriginal races, may be expected also to produce increased disease in the tracheobronchial tree.

Workers are by no means in agreement concerning the position of the lesions anterio-posteriorly. The majority favor the posterior or posterior-lateral walls and we are inclined to agree with this view, but there are also a large percentage of anterior and anterio-lateral ulcers. The minor difference may well be due to the position the patients assume during the late stages of the disease.

The apico-caudal localization is influenced somewhat by contiguity to large cavities which spread large numbers of bacilli up the bronchi. Many cases, however, seem to have random localization depending perhaps on the accidental penetration of the mucous glands or breaks in the epithelium wherein bacilli may enter.

The relation of tuberculous laryngitis to tracheobronchitis is no different than the relation of various other parts of the bronchial tree to each other. In about 75 per cent of the positive cases of the tracheo-bronchitis coming to autopsy there is a generalized involvement of all the structures. There are minor percentages, however, where the localizations are confined to only one or two of the other structures. Like tuberculosis in the gastro-intestinal tract, the point of greatest predilection seems to be at a point of stricture where the greatest activity and strain exists. In the air passages the larynx is the most vulnerable point especially in the region of the vocal cords or at the base of the epiglottis.

The question of pathogenesis seems to be more clearly defined than it was a few years ago. The majority of authors (as well as the preponderance of evidence) favors the direct implantation of bacilli, just as in tuberculous enteritis. The ruptured lymphatic nodes and lymphatic extension can generally only be thought of in the relatively rare cases of primary infection. There was no evidence in our series of the "back door" entrance of germs to the mucosa because the structure in and outside of the adventitious layers were free of disease. Equally rare is the hematogenous route of infection.

Finally a few suggestions may be made concerning the future

of the problem of tuberculous tracheobronchitis. Since it is now reasonable to assume that advanced pulmonary disease may develop tuberculous tracheobronchitis, and since most of the cases reveal the dangerous obstruction syndrome where treatment is limited to thoracoplasty, it follows that the earlier treatment is applied the better will be the result. If thoracoplasty is indicated, it should be done without hesitation, but for the cases who do not have such a good fortune there may be some relief in early use of streptomycin. While it is too early yet to evaluate the effect of streptomycin on all forms of pulmonary tuberculosis, there is already evidence that the drug clears up most lesions of the mucosa of the lower respiratory tract. Perhaps this new drug may hold the kep to the future of both the distressing obstructive tuberculous tracheobronchitis as well as the ordinary tuberculous lesions of the tract.

SUMMARY

1) A pathological study of tuberculous tracheobronchitis has been made on 667 cases coming to autopsy at the Chicago Municipal Tuberculosis Sanitarium.

2) Gross evidence of tuberculosis was found in 377 cases (56.5 per cent).

3) Microscopic study of a sample of grossly negative cases gave an estimated figure of 37.5 per cent of lesions in the remaining 290 cases, thereby increasing the positive findings of all cases to 72.0 per cent.

4) Counting only the 87.5 per cent of all far advanced lesions, it is observed that well over 80 per cent of far advanced cases have some degree of tuberculous tracheobronchitis.

5) The incidence was increased in the young age groups and in the female sex.

6) The pathogenesis seemed to be best explained by a direct implantation of bacilli in the mucous glands, or through breaches in the epithelium.

7) The location of lesions in the tracheobronchial tree was more or less random, although points of stress (larynx), posterior aspects of the tracheobronchial tree, and proximity to cavities seemed to be affected most.

8) The distribution of lesions was general in 242 cases (64.7 per cent); 65 (17.3 per cent) in lower portions of the tract; 19 (5.0 per cent) in trachea and left bronchus; 9 (2.4 per cent) in trachea and right bronchus; 31 (8.2 per cent) in trachea alone, and 11 (2.9 per cent) in bronchi alone.

9) The nature of the involvement was diffuse superficial infiltration in 12 cases (3.2 per cent); diffuse and deep infiltration and

infiltration and ulceration in 85 cases (22.8 per cent); circumscribed ulceration in 265 cases (70.3 per cent); fibroid stenosis in 5 cases (1.3 per cent); perforation in 6 cases (1.6 per cent), and caseous bronchitis in 4 cases (1.0 per cent).

10) There was microscopic evidence of specific tuberculous lesions in 88.1 per cent of the positive cases studied.

11) The hope for the future of tuberculous tracheobronchitis seems to be for early bronchoscopic study of all cases, early diagnosis, and early treatment of positive cases by thoracoplasty or streptomycin, or both.

RESUMEN

1) Se llevó a cabo un estudio patológico de la tráqueobronquitis tuberculosa en 667 casos autopsiados en el Sanatorio Municipal para Tuberculosos de Chicago.

2) Hubo hallazgos macroscópicos de tuberculosis en 377 casos (56.5 por ciento).

3) El estudio microscópico de una muestra de los casos que fueron negativos macroscópicamente dio una cifra calculada de 37.5 por ciento de lesiones en los 290 casos restantes, lo que aumentó los hallazgos positivos en todos los casos al 72.0 por ciento.

4) Si se cuenta solamente el 87.5 por ciento de todas las lesiones muy avanzadas, se observa que más del 80 por ciento de todos los casos muy avanzados presentan algún grado de tráqueobronquitis tuberculosa.

5) La frecuencia fue más elevada en los grupos etarios jóvenes y en el sexo femenino.

6) Parece que la mejor explicación de la patogenia es la implantación directa de los bacilos en las glándulas mucosas o a través de aperturas en el epitelio.

7) La ubicación de las lesiones en el árbol tráqueobronquial fue más o menos al azar, aunque los puntos de esfuerzo (la laringe), los aspectos posteriores del árbol tráqueobronquial y los lugares en la proximidad de cavernas parecían estar más afectados.

8) La distribución de las lesiones fue: general en 242 casos (64.7 por ciento); 65 (17.3 por ciento) en partes inferiores del aparato; 19 (5.0 por ciento) en la tráquea y el bronquio izquierdo; 9 (2.4 por ciento) en la tráquea y el bronquio derecho; 31 (8.2 por ciento) en la tráquea sola y 11 (2.9 por ciento) en los bronquios solos.

9) La naturaleza de la invasión fue infiltración superficial difusa en 12 casos (3.2 por ciento); infiltración difusa y profunda e infiltración y ulceración en 85 casos (22.8 por ciento); ulceración circunscrita en 265 casos (70.3 por ciento); estenosis fibrosa en 5 casos (1.3 por ciento); perforación en 6 casos (1.6 por ciento) y bronquitis caseosa en 4 casos (1.0 por ciento).

10) Hubo hallazgos microscópicos de lesiones tuberculosas específicas en 88.1 por ciento de los casos positivos estudiados.

11) La esperanza de la tráqueobronquitis tuberculosa en el futuro parece depender del estudio broncoscópico temprano de todos los casos, del diagnóstico oportuno y del tratamiento temprano de los casos positivos mediante la toracoplastia o con estreptomicina, o con ambos.

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Studies of Venous Pressure, Vital Capacity, Circulation Times and Electrocardiograms in the Course of Pulmonary Collapse Therapy*

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INTRODUCTION

This study was stimulated through the observation, by one of us (L.H.H.) of the appearance of the picture of right-sided heart failure in two patients who had been receiving artificial pneumothorax. Our purpose was to see if we could detect, early in the course of collapse therapy, any evidences of right heart strain. In addition to clinical observations we employed certain laboratory procedures which will be discussed below. Other similar studies have been carried out in this regard but as yet there has been nothing to demonstrate definite evidence of cardiac strain induced by collapse therapy.

Kroetz (quoted by Heise and Steidl[9]) in 1922 was apparently the first to study the venous pressure of patients before and after pneumothorax. Grellety-Bosirel (quoted by Heise and Steidl[9]) found that as a rule the establishment of pneumothorax produces no modifications of venous pressure. Gerdier (quoted by Heise and Steidl[9]) concluded that in pneumothorax venous pressure was a valuable indication of circulatory obstruction not always detected by clinical observation. He stated that venous pressure could predict that the collapse should be interrupted even when auscultation, x-ray studies and the respiratory rhythm failed to forecast disaster. However, others[1,9] have failed to note any significant changes of venous pressure during collapse therapy. Studies of the circulation time[1,6,7] have consistently shown a decrease with induction of collapse therapy but nothing to indicate right heart failure. Dock and Harrison[11] found in rabbits an initially increased volume flow of blood through the lungs and a delayed decreased volume flow. Electrocardiographic studies[3,6,10,14,16] have shown some changes which have been ascribed largely to rotation of the heart or the presence of air in the chest interfering with conduction of the heart current. In our present study we have

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employed all of these laboratory tests to determine the effect of pulmonary collapse on the circulation.

Method

The patients studied all had pulmonary tuberculosis of varying degree. A total of 32 patients were studied. They were classified in four groups based on the extent of parenchymal involvement as seen on the x-ray film. Groups I and II were based on the standards of the N.T.A.* classification. Group III did not follow this classification strictly because we felt that these patients with a cavity greater than 4 cm. in diameter would still remain in Group II providing there was not other parenchymal involvement to exceed the amount of lung tissue in one complete lung. Group III included those patients with involvement greater than the total tissue of one lung and Group IV included those patients with diffuse involvement of both lungs.

In the above groups there were thirty patients with artificial pneumothorax and two with unilateral phrenicotomy. All studies were carried out in a room at a uniform temperature of 26° C. The patient was allowed to rest for a full fifteen minutes before the tests were begun. The patient was first fluoroscoped and then vital capacity was determined using the McKesson apparatus and having the patient in an erect position.[7] The venous pressure was then determined in the antecubital veins bilaterally, using an 18-gauge needle and reading the venous pressure against a column of 4 per cent sodium citrate solution. The level of the vein was adjusted approximately to the level of the right atrium using the angle of Louis as a measuring point. Two per cent novocaine was injected intradermally at the site of insertion of the measuring needle. All pressures were determined in the left arm first and then in the right. With the right needle in place a mixture of five cubic centimeters of 20 per cent decholin solution and five minims of ether was injected rapidly into the vein. The patient was instructed to indicate immediately when the sensations of ether and a bitter taste were perceived. The responses were timed by stop watch. Electrocardiograms were then taken with the patient in a recumbent position. None of these patients had any elevation of temperature above 99° F. at the time of the tests. They were all thoroughly instructed in what to expect, thus eliminating fear. All procedures were carried out on the same day. Control studies were carried out about a week after the patient's admission to the hospital. Follow-up studies were then done one month and five months following the institution of collapse therapy.

*National Tuberculosis Association.

Results

Of the 32 patients, 12 were in Group I, 9 were in Group II, 7 were in Group III, and 4 were in Group IV, according to the above-mentioned classification.

Vital Capacity of the Lung

The study of the vital capacity in these patients revealed a progressive linear decrease with the increased degree of collapse. These findings are well illustrated in Table I. As can also be noted there apparently was no significant reduction in the control vital capacity of the lung until the class three and four cases were studied.

Venous Pressure and Circulation Times

In Table II we indicate the average circulation times and venous pressure ranges in the four classes of cases. It will be seen that there is a definite decrease in the circulation time in Group III and IV and definitely low venous pressure in Groups III and IV. The decrease in circulation time was accounted for chiefly by a changes in the pulmonary circulation time. The changes incident to the establishment of pulmonary collapse are noted in Table III. The degrees of increase or decrease are noted as measured against the average control values for each class. There was no significant

TABLE I

Class of Disease	1	2	3	4	All Groups
	Per cent	Per cent	Per cent	Per cent	Per cent
Average Control* Vital Capacity	96	87.3	82.2	71	84
Average Reduction in Vital Capacity One Month Following Institution of Collapse Therapy	76 or −20	63 or −24.3	64 or −18.2	53 or −18	64 or −20
Average Reduction in Vital Capacity Five Months Following Institution of Collapse Therapy	69 or −27	57 or −30.3	48 or −34.2	34 or −37	52 or −32

*Throughout this study the average has been calculated by simple arithmetical division of the total for all cases divided by the number of cases. The differences between the individual cases was so little that we felt this would be sufficient in determining the significance of our findings. The few instances in which there was greater deviation are discussed in the text of the paper.

difference in the venous pressure between the right and left arms either before or after collapse. In Groups I and III there was definite decrease in circulation time which seemed to be chiefly accounted for by a decrease in the ether time. This was much less marked in Groups III and IV, the possible explanation for which will be noted later.

It will be seen that the changes which occurred in circulation time at the end of one month of collapse therapy remained about the same at the end of five months, although in many instances the degree of collapse was greater at the end of five months.

Electrocardiographic Changes

In general the changes were not marked and certainly were not consistent with any definite evidence of cardiac strain. There was no apparent correlation of the degree of pulmonary collapse, extent of the disease, the degree of mediastinal shift or the side of the collapse with any specific change in the tracings. Alterations in venous pressure or circulation time also had no consistent bearing on the appearance of the cardiogram.

TABLE II

Circulation Time

Class	1	2	3	4
Ether	7.5 sec.	9.0 sec.	6.2 sec.	6.3 sec.
Decholin	13.5 sec.	13.0 sec.	12.4 sec.	12.1 sec.
Venous Pressure	8 cm.	4.9 cm.	4.1 cm.	4.1 cm.

TABLE III

Average Change in Circulation Time

Class		1	2	3	4	All
Ether	1 mo.	−1.2 sec.	−3.2 sec.	−1.0 sec.	−0.8 sec.	−1.6 sec.
Ether	5 mo.	−0.6 sec.	−2.4 sec.	−0.7 sec.	−1.1 sec.	−1.2 sec.
Decholin	1 mo.	−2.1 sec.	−3.2 sec.	+0.7 sec.	−1.1 sec.	−1.4 sec.
Decholin	5 mo.	−1.0 sec.	−2.7 sec.	−0.2 sec.	−0.6 sec.	−1.1 sec.

Average Change in Venous Pressure

	1 mo.	+0.8 cm.	+0.2 cm.	+1.4 cm.	No Change	+0.6 cm.
	5 mo.	−1.0 cm.	−1.2 cm.	−0.5 cm.	No Change	−0.7 cm.

The average cardiac rate in these 32 cases was 88 per minute. Eleven of them were below 80 and some of these had far advanced disease. At the end of one month of collapse therapy there was uniform increase in the heart rate. At the end of five months, 13 patients still had increased heart rates, 7 were decreased, and 15 were unchanged. Again there seemed to be no correlation between the changes in heart rate and the stage of the disease. None of the patients developed any arrhythmia. Four cases showed development of some right axis deviation. This was most pronounced in a patient who had a minimal mitral lesion in addition to his tuberculosis. He has been receiving pneumothorax now for 16 months and has shown no clinical evidence of right heart strain. In three of these cases the pneumothorax was on the right side. In one case it was bilateral.

The most consistent changes observed at the end of one month of institution of collapse therapy were:

1. Decrease in the voltage of the R wave in 17 cases in Lead I and in 16 cases in Lead IV-F;
2. Decrease of the T wave in 15 cases in Lead I and in 18 cases in Lead IV-F;
3. In 9 cases, a decrease of T wave in Lead II;
4. In 10 cases, an increase of the R wave in Lead III which was accompanied in 5 cases by an increase in the S wave of Lead I;
5. In 6 cases, a decrease of the T wave in Lead III;
6. No change in 4 cases.

At the end of five months no further consistent changes were observed. An interesting observation was that the changes in Lead I seemed to be mirrored by the changes in Lead IV-F.

Discussion of Results

As stated at the outset of this paper, we attempted to demonstrate early evidence of right heart strain in the institution of collapse therapy. As can be gleaned from our results there is no evidence of right heart failure so far as these cases have been followed by the procedures herein utilized. It may be added that in no case was there clinical evidence of congestive failure, although some of the patients complained of dyspnea.

Studies of the vital capacity revealed an almost linear reduction with the increase of pulmonary collapse. This is quite obvious since the vital capacity is dependent on the volume of respiratory surface. This finding has also been pointed out by Stewart and Bailey[6] and Feinsilver.[1] We feel that the determination of the vital capacity is of little value in determining the presence of involvement of pulmonary tissue unless this is rather extensive. Apparently the uninvolved portions of lung can compensate greatly until so much

lung is involved as in the Group III and IV cases that there is a significant diminution in respiratory reserve with consequent reduction in vital capacity. These findings are corroborated by others.[2]

On the whole, these patients in their control studies exhibited normal circulation times, using 13 seconds for the decholin time and 7 seconds for the ether time as normals. There were occasional patients in whom the circulation time seemed to be a little prolonged but there were no particular factors with which to correlate these findings. The circulation times in the Groups III and IV patients seemed to be more rapid. The fact that this was chiefly accounted for by a decrease in the ether time would suggest that extent of involvement of the pulmonary tissue may have sufficiently diminished the size of the vascular bed to account for the above reduction. The circulation times were also uniformly decreased with collapse therapy. This seemed to be most marked during the first month and less at the end of five months. The fact that this was less marked in the Group III and IV cases was probably due to the greater difficulties in obtaining good collapse in these cases. The changes again were due principally to reduction in the ether time and again we explain the changes through a reduction in the pulmonary vascular bed. The fact that these changes are less marked at the end of five months in view of increased pulmonary collapse can be explained only on the basis of change in heart rate since at the end of one month the heart rates were uniformly increased over the control levels, whereas at the end of five months in only less than half of the cases was there still an increase in heart rate. Feinsilver[1] has noted essentially similar findings. Others[6,7] have noted very little change in the circulation times.

Venous pressure readings in the controls were essentially normal. They tended to be generally lower in the Group III and IV cases. It has been pointed out[13] that there is some evidence of emaciation being a factor in lowering of venous pressure. This is the only factor we could evolve as an explanation for the above since such things as vis-a-tergo or intrathoracic pressure could not be demonstrated to be essentially different in the Group III and IV and in the Group I and II cases. With collapse therapy, there was in general a slight rise in venous pressure at first but this promptly subsided. These changes were not large enough to be considered of any significance. In one of the Group III cases, there was a significant rise of venous pressure bilaterally, (3 cm.), which was maintained at the end of five months. However, we could demonstrate no evidence of right heart failure to explain this. There was one other patient in Group I who had a rise in venous pressure of 3 cm. bilaterally at the end of one month but this promptly

returned to normal levels at the end of five months. There was no mediastinal shift or any particular adhesions to explain the temporary but definite rise in this case. It has been found by others[1,6,9] that there were no significant changes in venous pressure incident to collapse therapy. We noted that the degree of mediastinal shift has no particular effect on the venous pressure. A few cases with marked pleural effusion and pronounced mediastinal shift showed little or no alteration in the venous pressure.

Hurst and Brand[8] have pointed out that high tension pneumothorax may cause a rise in venous pressure on the same side, with or without mediastinal shift. They feel that venous pressure changes occur on the side of the more extensive tuberculous lesion or collapse measure and explain it on the basis of local obstruction of the subclavian or innominate vein. This phenomenon did not occur in our studies so far as we have carried them. It is not surprising that we did not observe more changes indicative of increased tension in the pulmonary circuit since it has been pointed out[12] that the vascular bed of the lungs must be reduced by about 60 per cent before the pressure is increased in the lesser circulation.

The electrocardiographic studies revealed even less evidence of any cardiac strain than did the above discussed procedures. As previously mentioned the most consistent findings were decrease of the R and T waves in Lead I and IV-F. As Master[14] has suggested the low voltage of the Q-R-S complex and the T waves is probably to be accounted for either by the rotation of the heart or by the fact that air in the chest is a poor conductor for the heart current. We are inclined to lean to the latter explanation since in these cases we were not able to demonstrate actual cardiac rotation fluoroscopically. In four patients there was development of right axis deviation, the pneumothorax being on the right side in three cases and bilateral in one case. In Master's[14] cases all of the patients with right pneumothorax showed a right axis deviation of the Q-R-S complex. He feels that in right pneumothorax particularly that a rotation of the heart occurs and probably accounts for the above. He followed two cases who showed disappearance of the above changes with disappearance of the pneumothorax. His study, however, was concerned with only seven cases, five of which were spontaneous pneumothorax. He found the most marked changes with displacement of the mediastinum. We could not conclude that in one case with marked mediastinal shift that any greater change was noted in the electrocardiogram. Anderson[3] found that neither the degree of pulmonary collapse maintained nor the duration of the collapse therapy had any definite relationship to the form of the electrocardiogram. Again his study was different from ours in that he did not compare the

electrocardiograms of the same patient before and after pneumothorax. Hansen and King[16] felt that the change observed, most frequently in the R and T waves, were due to change in position and not myocardial damage. Similar conclusions have been reached by others.[6] Boas and Mann[15] feel however that displacement of the heart in their experience had no effect on the result of the electrocardiograph. Our studies seemed to corroborate the latter statement. Simon and Baum[10] consider the presence of adhesions as important in producing predominance of either ventricle. This could not be demonstrated in our series. It appears that in those cases in which axis deviation appeared that it was due to rotation of the heart. The change in the R and T waves was most likely on the basis of presence of air in the chest.

SUMMARY

Simultaneous studies of the venous pressure, circulation time, vital capacity of the lung, extent of pulmonary involvement, and electrocardiograms have been made in a series of 32 cases with pulmonary tuberculosis.

These studies were carried out as controls and again at the end of one month and five months after institution of collapse therapy. In 30 cases collapse was induced by artificial pneumothorax and in two cases by phrenicotomy. Factors of emotion, environmental temperature, and body temperature were noted and controlled. The control studies were carried out after the patient had been in the hospital for a week.

The purpose of the study was to try to demonstrate any evidence of right heart failure early in the course of collapse therapy. These patients were observed clinically as well as through the laboratory.

CONCLUSIONS

1) In no case was there clinical or laboratory evidence of right heart strain.

2) Decrease of vital capacity of the lung is directly proportional to the degree of collapse. Vital capacity does not seem to be materially affected in the controls until there is extensive pulmonary involvement.

3) The venous pressure is not significantly affected by pneumothorax or phrenicotomy.

4) The circulation times are decreased initially by pulmonary collapse chiefly due to a decrease in the ether time. It is suggested that this results from a decrease in the pulmonary vascular bed and an increase in heart rate.

5) The electrocardiogram has shown no evidence indicative of

right heart strain. The changes noted apparently are due to rotation of the heart and presence of air in the chest.

6) Only through more prolonged studies of these patients, may it be possible to detect any changes in the procedures herein employed which may indicate evidence of right heart failure before frank clinical features appear.

RESUMEN

En una serie de 32 casos de tuberculosis pulmonar se han llevado a cabo estudios simultáneos de la presión venosa, velocidad de circulación, capacidad vital del pulmón, extensión de la invasión pulmonar y electrocardiografías.

Se hicieron estos estudios, primero para que sirvieran de pauta, y se repitieron después de un mes y después de cinco meses de haberse iniciado la colapsoterapia. En 30 casos la forma de colapso empleada fue el neumotórax artificial, y en dos casos la frenicotomía. Se tomó en cuenta y se corrigió el factor emocional y, también, la temperatura del ambiente y del cuerpo. Se llevaron a cabo los estudios reguladores después de que el paciente había estado una semana en el hospital.

El propósito del estudio fue el de tratar de demostrar tempranamente en el curso de la colapsoterapia signos de insuficiencia del corazón derecho. Se observó a estos pacientes tanto clínicamente como con exámenes de laboratorio.

CONCLUSIONES

1) En ningún caso hubo signos clínicos o de laboratorio perjudiciales al corazón derecho.

2) La disminución de la capacidad vital del pulmón es directamente proporcional al grado de colapso. No pareció ser afectada notablemente la capacidad vital de los reguladores sino cuando existía invasión pulmonar extensa.

3) Ni el neumotórax ni la frenicotomía afectan importantemente la presión venosa.

4) El colapso pulmonar causa inicialmente una disminución en el tiempo de circulación debida, principalmente, a disminución del tiempo del éter. Se supone que esto es debido a disminución en la red vascular pulmonar y a aumento en el número de latidos del corazón.

5) La electrocardiografía no ha revelado ningún signo indicativo de daño al corazón derecho. Las alteraciones observadas se deben aparentemente a la rotación del corazón y a la presencia de aire en el tórax.

6) Sólo mediante estudios más prolongados de estos pacientes

podría ser posible descubrir en los procedimientos aquí empleados alteraciones que pudieran indicar insuficiencia del corazón derecho antes de que aparezcan francos signos clínicos.

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Right Heart Failure After Thoracoplasty*

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From a study of the available literature it would appear that little attention has been paid to the question of cardio-pulmonary insufficiency after long-standing thoracoplasty. In the Scandinavian literature[1] two cases are reported in which death occurred three and four years respectively after thoracoplasty with necropsy findings of distinct right cardiac hypertrophy and dilatation. In a recent article on chronic cor pulmonale[2] one case is mentioned in which marked right ventricular hypertrophy was found at necropsy, nine years after a thoracoplasty. The authors state "undoubtedly, the question of whether or not long-standing thoracoplasty may result in a strain on the right side of the heart is a subject which deserves further study." We recently had occasion to observe two cases of cardio-pulmonary insufficiency after long-standing thoracoplasty.

CASE REPORTS

Case 1: I. C., White, female, 39 years old. Diagnosis of pulmonary tuberculosis in 1934. Pneumothorax on the right instituted in June 1936. Permanent phrenic paralysis performed in July 1936. Tuberculous empyema with bronchopleural fistula developed in 1939. Patient was admitted to the National Jewish Hospital and a thoracotomy followed by a three stage ten rib thoracoplasty and Schede procedure was performed. Vital capacity which was found to be 36 per cent before the operation was 26 per cent after surgical procedure. After discharge from the institution patient was seen in the Out-Patient Department at regular intervals. She presented no complaints except for slowly increasing dyspnea on exertion. In the last week of May 1946 she developed what seemed to be an upper respiratory infection with slight temperature elevation and nonproductive cough. There was severe increasing dyspnea despite routine treatment of the upper respiratory infection and she was readmitted to the hospital. On examination she was found to be dyspneic, orthopneic and cyanotic. Tachycardia with a pulse rate up to 140 was present. Blood pressure which had been normal previously was elevated to 170 systolic and 108 diastolic. Slight distension of the neck veins was noted and tenderness to pressure was found over the right upper quadrant. X-ray film showed cardiac enlargement by comparison with previous films. EKG gave evidence of slight right axis deviation. Venous pressure studies were done and showed elevation to 21 and 24.5 cm. respectively on the right and left side with positive response to right upper quadrant pressure (Pasteur-Rondot test[3]). She was placed on bedrest and given a full course of digitalization with a purified digitalis

*From the National Jewish Hospital at Denver.

product. Her dyspnea subsided gradually with disappearance of the other chemical findings and she was discharged after three weeks. At the time of discharge, x-ray film showed reduction of her heart size, venous pressures were in normal range (R.A. 5 and L.A. 9) and the modified Pasteur-Rondot test was negative. She was given a maintenance dose of digitalis and advised to move to a lower altitude. According to reports received from her since, she has had no further episodes of cardiac decompensation and her exertional dyspnea has been reduced.

Case 2: P. M., White, female, 46 years old. Onset of pulmonary tuberculosis in 1922 with hemoptysis. Left pneumothorax was induced and permanent left phrenic paralysis performed in 1930. Because of evidence of cavity in the left upper lobe and positive sputum she was re-admitted in 1935 and a four rib thoracoplasty on the left was performed. She was followed in the Out-Patient Department and presented no complaints except gradually increasing dyspnea on exertion. In February 1946 she was seen in the Out-Patient Department with indefinite gastro-intestinal symptoms. Gall bladder and gastro-intestinal x-ray series were negative. Chest x-ray film showed definite cardiac dilatation; EKG, right axis deviation with signs of right ventricular strain. She was not seen again until August 1946 when she was first admitted to a general hospital in full congestive failure. She was digitalized and after a short stay was re-admitted to the National Jewish Hospital. On admission she was markedly dyspneic and cyanotic, with moist rales over the bases, liver enlargement and ankle edema. Venous pressures were: R.A. 12, L.A. 16½; right upper quadrant pressure resulted in venous pressures of R.A. 18, L.A. 23. EKG showed right ventricular strain and digitalis effect, x-ray film gave evidence of marked cardiac enlargement. Her digitalization was continued and oxygen was given by nasal catheter. Her condition improved with reduction of edema and weight. Venous pressures returned to normal, R.A. 6 and L.A. 9.5. When oxygen was discontinued, her edema recurred and despite low sodium-acid ash diet and intensive use of mercurial diuretics she showed increasing fluid retention. Oxygen was again begun and after a time she was discharged home where she continued on the same routine with no improvement in her condition. She died in congestive failure in October 1946. No necropsy was done.

Discussion and Comment

From a clinical point of view the two above described cases illustrate the importance of early recognition of the cardiac factor in patients developing cardio-pulmonary insufficiency in chronic pulmonary tuberculosis with permanent collapse procedures. The anoxemia caused by the ventilatory-respiratory insufficiency, results in dyspnea and cyanosis and the superimposed cardiac involvement can be recognized "early" only if attention is paid to "specific" cardiac signs of right heart failure. If only suggestive evidence is found, the patient should then be subjected to a thorough diagnostic work-up, including x-ray, electrocardiogram, and venous pressure studies. Our observations on these two and other cases of cardio-circulatory failure in chronic pulmonary disease have led us to endorse Spain and Handler's[2] impression that routine cardiac treatment, especially digitalization, seems to

be of value only if applied early. We also believe that oxygen should be administered early so as to prevent irreversible changes in the myocardium as well as pulmonary edema.

As to the causes of right heart failure after thoracoplasty there is a wide field for speculation. Bruce[1] feels that the marked kyphoskoliosis developing in some cases of thoracoplasty may be an important factor. He states "the plastic patient often shows a striking gross anatomic resemblance to the kyphoscoliotic patient." As attractive as this theory is, in neither of our cases was there sufficient deformity to consider it of etiological significance.

Studies of the respiratory function of lungs before and after thoracoplasty by bronchospirometry as those by Pinner, Leiner, and Zavod[4] tend to prove that respiratory function is not unfavorably influenced by thoracoplasty. However, they state that this does not apply to thoracoplasties with pre-existing or added diaphragmatic paralysis. This was present in our two cases and probably contributed to the pulmonary insufficiency. It is necessary to point out that even temporary phrenic crush may result in permanent paralysis or at least partial palsy. From our own experience we feel that this measure contributes greatly to impairment of cardiopulmonary reserve.

Kaltreider, Fray and Phillips[5] in an interesting study of pulmonary-ventilatory and respiratory function as well as function of the cardiovascular apparatus came to the conclusion that surgical interference with the chest-cage results in an anoxic anoxemia. The average value for the oxygen content of the arterial blood was below the average normal value. However, frank signs of right ventricular failure were not noted in their series and they state "this is not surprising when one realized that the vascular bed of the lungs must be reduced by approximately 60 per cent before the pressure is increased in the lesser circulation." They make the additional statement: "there are however, other factors, such as anoxemia of the myocardium which may put an additional load on the right ventricle in patients with thoracoplasty."

Cardiac failure in chronic pulmonary tuberculosis with permanent collapse procedures is obviously due to a combination of factors. The collapsed pulmonary area is usually not large enough to cause persistent pulmonary hypertension and its sequel, cor pulmonale. Additional factors which have been considered such as rigidity of the chest wall which interferes with normal respiratory-circulatory cycle, compensatory emphysema of the uncollapsed lung parenchyma of the thoracoplasty lung, capillary congestion and emphysema of the contralateral lung may each be contributory. The result of combining any of these factors appears to be anoxic anoxemia resulting in an added burden to

the myocardium. This chronic anoxemia as well as obliteration of part of the pulmonary capillary bed plus additional factors of age, coronary artery disease, or intercurrent upper respiratory infection will result in heart failure.

Obviously no definite causal relationship can be claimed to exist between the permanent collapse procedure performed in the two presented cases and the occurrence of right heart failure. However, by prolonging the life expectancy of individuals with chronic pulmonary tuberculosis by thoracoplasty, we may be presented with an increasing incidence of cardiac failure. It is definitely felt that patients with chronic pulmonary disease with or without collapse procedure who are dyspnoeic should be enjoined to live at low altitudes to avoid any additional strain upon the myocardium.

SUMMARY

1) We have presented two cases of right heart failure with chronic pulmonary tuberculosis with associated thoracoplasty and permanent phrenic paralysis.

2) While the theoretical considerations for such an association have been given, no definite causal relationship could be postulated.

3) Regular cardiovascular examinations were stressed for patients with increasing dyspnoea who had undergone permanent collapse procedures. Early treatment in cases of right heart failure appeared of definite value in restoring compensation as opposed to late cases.

RESUMEN

1) Hemos presentado dos casos de insuficiencia del corazón derecho con tuberculosis pulmonar crónica, asociada con toracoplastia y parálisis permanente del nervio frénico.

2) Aunque se han dado las razones teóricas de tal asociación, no se pudo enunciar ninguna relación causal bien definida.

3) Se recalcó la importancia de llevar a cabo exámenes cardiovasculares metódicos en pacientes con disnea creciente que habían sido sometidos a procedimientos permanentes de colapso. El tratamiento temprano de los casos de insuficiencia del corazón derecho pareció ser de valor bien definido para restablecer la función cardíaca, en contraste a los casos tratados tardíamente.

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The Synergism Between Mycotic and Tuberculous Infections of the Lungs*

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INTRODUCTION

The idea of a commensal relationship between fungus and tuberculous infections of the lungs has been suggested in the past. It is my impression that the importance of their coexistence has not been fully appreciated by the medical profession. The prevalent knowledge that fungi are ordinarily saprophytic and ubiquitous has, perhaps, led us into the erroneous attitude that all fungi found in the sputum are harmless contaminants, and their importance as a factor in an existing tuberculous process is slight and of no consequence in the consideration of the subsequent course of the tuberculous invalid who is unfortunate enough to be the host to both a tuberculous and a fungus infection. My object in this paper is to refute the impression that these coexisting fungi are harmless, saprophytic invaders, and to advance added data to support the premise that an infection of the lungs with pathogenic fungi in association with a tuberculous infection of the lungs is not harmless and coincidental, but that it definately increases the activity and virulence of the tuberculous process. An attempt will be made to analyze the possible ways in which this activation or modification takes place. Furthermore, it is hoped that a free discussion at this meeting may add to our knowledge of the underlying reasons for this increased invasiveness of the mycobacterium tuberculosis when a mycotic infection is also present; especially valuable will be observations along the trends of bacteriological, biochemical and immunological analysis.

The Problem of Diagnosis

The detection of a fungus infection with, or without, the presence of pulmonary tuberculosis may be fraught with difficulty. The symptoms and clinical course of pure pulmonary mycoses may be identical with those of pulmonary tuberculosis. Even the roentgenogram may be of no differential value. It is well to remember that fungi found in the expectorated sputa are usually saprophytic; especially is this true of the aspergillus, penicillium,

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and the saccharomyces. It has been affirmed that the aspergilli and the penicilia are three times more frequently found than all other species combined. It has been my personal experience that the saccharomyces are found saprophytically in the sputum more commonly than both the aspergillium and the penicillium species. There is only one certain way to guard against the inclusion of saprophytic fungi, and that is by obtaining the specimen for examination by intratracheal aspiration, cultivation on Sabouraud's medium, and animal inoculation to prove the pathogenicity of the fungus. The problem is also enhanced by the relationship of certain fungi, the actinomyces, to the mycobacteria. Most bacteriologists[1] classify both the mycobacteriaceae and the actinomycetaceae under the order actinomycetales, and identify them as bacteria (schizomycetes). There is no doubt that the actinomycetes are phylogenetically very closely related to the mycobacteria tuberculosis. Certain acid-fast forms of actinomycetes bear a close resemblance to the tubercle bacilli in morphology, cultural characteristics, pathogenicity and immunity reactions. The actinomycetes are very labile and prone to change their morphology, and some species may at least temporarily fail to form a mycelium and grow entirely in a bacillus-like form.

Common Forms of Fungi Observed

The pathogenic fungi, found in association with pulmonary tuberculosis in our studies[2] of the problems were, the cryptococcus hominis, monilia bronchialis, saccharomyces hominis, aspergillus fumigatus, sterigmatocystis nidulans, and sporotrichum schencki. The penicillium fungus was never encountered even as a probable saprophyte in the expectorated sputum. The fungi, listed above, comprise the results in 12 cases of proved tuberculosis in whom we succeeded in isolating the fungi, by intratracheal aspiration, in pure culture, followed by animal inoculations in which the fungi were recovered from the animals' lung tissues. In 8 other patients, in whose sputum tubercle bacilli were absent, fungi were similarly isolated, and studied with like results. Five of these cases were pure bronchomycoses, including 3 cases of sporotrichosis, one aspergillosis, one cryptococcosis. One bronchiogenic carcinoma was found in association with monoliasis. There were 27 patients, whose expectorated sputa contained fungi, which we were not able to obtain from the trachea. We classified them as saprophytes. They were identified as 2 cases of cryptococcus Kützing, 2 monilia Persoon, 2 sporotrichum Link, 2 acremonium Link, 2 aspergillus Micheli and 17 cases of saccharomyces Meyen.

There has been a gradual, but progressive, evolution in the conviction among physicians, during the past decade, that pul-

monary mycoses, associated with pulmonary tuberculosis, must be searched for and seriously considered in every patient with pulmonary tuberculosis. Dodge[3] reviewed the literature up to 1933, and found only 26 cases of pulmonary mycoses, which either simulated pulmonary tuberculosis, or were in association with a tuberculous infection of the lungs. This would indicate, considering my personal experience, that the relationship of the two diseases was not being seriously considered, and the fungus organisms were not being diligently sought for in the sputa of tuberculous patients before that time. Dodge found 19 cases of filamentous fungi, listed as 9 cases of nocardia, 2 oidium, 2 sporotrichum, 2 penicillium, 2 aspergillus, and 1 sterigmatocystis. There were found 7 cases of yeast-like fungi, diagnosed as 4 cases of cryptococcus, 2 monilia, and 2 endomyces. It is to be noted that of Dodge's 19 cases there were 13 of the classification of fungi imperfecti, and of our 18 cases of proved pulmonary mycoses, with and without tuberculosis, 10 of the fungi were fungi imperfecti. It would seem logical that we may expect fungi imperfecti to be found in two-thirds of the mycotic infections of the lungs. Other recent reports also indicate this to be true.

Previous Observations

Mary Lapham[4] wrote in 1926, "There are cases of aspergillosis on record which subsequently became tuberculous, and Renon says that this is a dangerous feature of the disease. While it is not generally believed that the development of tuberculosis is in any way associated with a disease so rare that it is a curiosity, how do we know that this disease is so infrequent? Would it be strange if such a disease should seriously complicate, or even inhibit recovery in a case of tuberculosis?" She subsequently stated that when a case of tuberculosis is complicated by aspergillosis, recovery seems more difficult and more unstable because there is a decided tendency to relapses. Also, Castellani[5] reported that some of these mycotic infections resemble tuberculosis clinically, and that the physical and roentgenologic findings will not aid in differentiating them from tuberculosis. Numerous observers have noted the malign effects the presence of a fungus infection has upon the course of pulmonary tuberculosis. This fact seems established firmly in the minds of competent clinicians.

Symbiotic and Synergistic Considerations

A consideration of the symbiotic relationships between mycobacteriae tuberculosis and fungi leads us into the realm of speculation, diverse viewpoints, and lack of definite knowledge. Perhaps one may seem bold, indeed, who essays to piece together the

available knowledge, and add purely theoretical considerations of his own, dealing with the symbiotic and synergistic problems of mycotic and bacterial association as they pertain to the problem before us today. The term "symbiosis" will be used in the same sense as employed in general biology, rather than in the more restricted conception of bacteriologists. In the latter meaning symbiosis refers to the growth of certain bacteria only in the presence of certain other bacteria.

The theories of Professor Wallin[6] embody clearcut facts of symbiosis in the sense to be used in our discussion. He has proposed the term "prototaxis" to express a principle, defined by him, as "the innate tendency of one organism or cell to react in a definite manner to another organism or cell," and "positive prototaxis," as "the affinity of one organism or cell for another organism or cell." The term "symbiosis," then, indicates mutual benefit is derived; "parasitism," on the contrary, presupposes some benefit to the parasite with either a harmful or no response on the part of the host. Wallin further affirms "symbiosis is applied to those cases where mutual benefit is derived. It is evident that there is no sharp distinction between parasitism and symbiosis considered from the point of view of prototaxis; parasitism and symbiosis are merely different end responses in the expression of one and the same biological principle."

We have no proof that there is any symbiotic relationship between tubercle bacilli and pathogenic fungi when they are subjected to the same cytoplasmic environment in the lung tissues. We know they are subjected to the same encompassing factors—thermal, photic, electrical, chemical, and physical. It may be probable that in combined fungus and tuberculous infections of the lungs that the tuberculous implantation may be the primary one, and that the fungi, attacking the lungs later, produce some change, chemical or physical, which reinactivates tubercle bacilli, which have become less virulent because of localizing and focalizing tissue processes and specific immunologic influences. Recently, reports from the Edward J. Meyer Memorial Hospital and the Department of Medicine, and the University of Buffalo School of Medicine,[7] stated that subcultures of a mold of the penicillium group, obtained from a contaminated culture of tubercle bacilli, showed rapid and luxuriant growth on other cultures of tubercle bacilli at room temperature, that the mold grew faster and sporulated earlier than it did on similar sterile media. Their further experiments indicated the mold inhibited the growth of tubercle bacilli in culture, and, also inactivated tuberculin in two hours. This tendency for molds in inactivate tuberculin, that show bacteriostatic activity against tubercle bacilli in the test tube, had

been previously observed by others. In these test tube experiments, although no growth of tubercle bacilli was observed, acid-fast bacilli were still present in smears after several months incubation of the tubes. Could these findings suggest the mold, when in association with tubercle bacilli in the lungs, inhibits the building up of specific immunity, local and systemic, on the part of the tubercle bacilli? The conditions within the tissues of the lungs are vastly different than in the laboratory test tube. In the former it is quite unlikely the implantation of tubercle bacilli and fungi occur at the same, identical time, as was the case with the inoculation of the test tubes. One or the other, tubercle bacilli or fungi, would be expected to have had prior existence for weeks, months, or years, in the lung tissues, and to have become firmly engrafted with their pathological processes before the other one infected the individual.

One might reasonably expect biochemical interchanges to be produced when pathogenic fungi and tubercle bacilli both infect the pulmonary tissues. Tubercle bacilli usually become less virulent, and assume a less active and invasive state after residing within the lung tissues for varying periods of time. This lessened activity is dependent upon such factors as the initial virulence of the tubercle bacillus and its reaction to general bodily resistance, the acquired localizing tissue defense mechanisms and systemic immunologic developments, as well as the lessened nutrition for the bacilli within the tissues, which may partly or wholly encapsulate them. Such bacilli, deprived of an adequate, or balanced nutrition are less capable of multiplying because their limited intracellular reserve foodstuffs are thereby diminished. As a consequence there will be a marked reduction in the metabolic activities of the tubercle bacilli. It has been proved that inactive, or resting, bacteria regain their catabolic activities when any substance, normally utilized in their catabolism, is rendered available to them. The possibility that the mycelial fungi, especially, may supply nitrogen, at least, is suggested by our knowledge of the effects of certain mycelial fungi in the metabolism, life history, and successful development of higher plants. Mycelial fungi grow in and on the roots of many of these plants; the legumes, for example, are furnished available nitrogen by this means. The fungus mycelium may also serve to increase the decomposition of the infected tissues, thereby the cytoplasmic resistance to the inroads of the tuberculous infection is decreased. The highly speculative nature of these remarks is apparent to me. They are advanced as ideas of no definite value, perhaps, and solely for their importance in bringing about a more plausible explanation from your discussion of this problem.

In some cases of pulmonary mycoses the pathology closely resembles tuberculosis. Surrounding the fungus infection there is usually tissue destruction and softening, an accumulation of leucocytes, and the formation of pus. The abscesses become surrounded with a dense layer of new fibrous tissue, infiltrated with mononuclear leucocytes, and sometimes containing giant cells. This is interesting to note, even though no relationship is suggested, when we remember the frequently observed similarity in the clinical courses of chronic pulmonary tuberculosis and pulmonary mycoses.

Previous Personal Observations

In a former paper,[2] written with Dr. H. N. Gemoets, our findings were reviewed of an investigation of 301 consecutive admissions to the Houston Tuberculosis Hospital. The object of the survey was to determine the degree, if any, of association of mycotic infections in tuberculous patients. Two hundred and ninety-five of the patients were definitely tuberculous; the remaining 6 cases were repeatedly sputum negative for acid-fast bacilli. In 45 patients of the total number (301) we isolated fungi from the expectorated sputum, but in only 18 cases did we obtain the fungi by means of an intratracheal aspiration. The remaining 27 cases, comprising the group in which we were unable to recover the fungi from the trachea, were excluded from our study because of the likelihood of the fungi, obtained from the mouth, being saprophytic. The fungi from the 18 patients were subjected to intensive studies, including injection into the lungs of rabbits. In every one of the cases pathological changes were noted in the lung tissues of the inoculated rabbits, and the identical fungus was recovered, again, by culture. We believe we were justified in deciding that these fungi were pathogenic. The larger number of these fungi were filamentous in type. We were greatly surprised at the remarkably short period of time elapsing between the inception of clinical symptoms in these patients and the development of advanced pulmonary disease. In 8 (77 per cent) of our 12 proved tuberculous patients the average time since the onset of their primary clinical symptoms was just 9.6 months. However, in this short period the lung changes had become far advanced. We suspected there was some cooperative influence at work to bring about such devastating changes in less than 10 months duration of known symptoms of sickness.

SUMMARY

An attempt has been made to discuss the commensal relationship between fungus and tuberculous infections of the lungs. The

coexistence of these two infections produces a more rapidly fatal issue in tuberculous individuals than one should expect from a consideration of the duration of the disease process in the lungs. Fungi from the expectorated sputum are usually saprophytic. It is safest to obtain the sputum specimens by intratracheal aspiration, and subject the fungus to animal inoculation, if there is any doubt regarding its pathogenicity.

Physicians should search diligently for coexisting pathogenic fungi in every tuberculous patient whose clinical course is unusually rapid or prone to relapse. The fact that 1.6 per cent (5 patients) of our clinically diagnosed tuberculous patients harbored pathogenic fungi in their tracheal excretions, although tubercle bacilli were never recovered from these 5 patients, should be remembered. The mycelial fungi are found more frequently than yeast-like fungi as the etiologic agents of pulmonary mycoses, associated with pulmonary tuberculosis. The close phylogenetical relationship of the mycobacterium tuberculosis to certain fungi, the actinomyces, and the frequently noted similar pathological changes in mycotic and tuberculous disease of the lungs are of interest. All ideas dealing with the cooperative relationship between the tubercle bacilli and fungi existing together in diseased tissues are highly speculative, as little, if anything, is known regarding such interacting or interplaying processes. Our investigations would suggest, in a limited number of cases, that the association of a fungus infection of the lungs in a patient having pulmonary tuberculosis would augment and accelerate the activity of the tubercle bacilli, either directly, or by lowering the vitality and resistance of the patient to the end that a more unfavorable course of the tuberculous disease, marked by relapses and relatively rapid progression, may be observed. It is suggested that there is some factor present in this interplay of two coexisting infectious diseases, some prototaxic or symbiotic influences, which causes an added infection of the lungs with pathogenic fungi to increase the ravages of pulmonary tuberculosis.

RESUMEN

Se ha intentado discutir las vinculaciones entre las infecciones fúngicas y tuberculosas de los pulmones. La coexistencia de estas dos infecciones en individuos tuberculosos produce un desenlace más rápidamente fatal de lo que uno esperaría tomando por base la duración del proceso morboso en los pulmones. Los hongos obtenidos en el esputo expectorado generalmente son saprofitos. Es mejor obtener los espécimenes de esputo mediante la aspiración intratraqueal e inocular los hongos en animales, si existe alguna duda en cuanto a su patogenicidad.

Los médicos deben buscar asíduamente hongos patógenos coexistentes en todo paciente tuberculoso cuyo curso clínico es excepcionalmente rápido o propenso a recaídas. Debe recordarse el hecho de que el 1.6 por ciento (5 pacientes) de nuestros pacientes tuberculosos diagnosticados clínicamente tenían hongos patógenos en sus excreciones traqueales, aunque nunca se pudo encontrar bacilos tuberculosos en estos 5 pacientes. Se encuentran más frecuentemente los hongos micelianos que los hongos parecidos a la levadura, como agentes etiológicos de micosis pulmonares asociadas con tuberculosis pulmonar. La estrecha relación filogenésica entre el microbacterio tuberculosis y ciertos hongos, los actinomicetos, y las alteraciones patológicas semejantes, frecuentemente encontradas en enfermedades micóticas y tuberculosas de los pulmones, son de mucho interés. Todas las ideas tocantes a la relación cooperativa entre el bacilo tuberculoso y los hongos, cuando existen juntos en tejidos patológicos, son altamente especulativas, ya que poco o nada se sabe de la acción recíproca de tales procesos. En un número limitado de casos, nuestra investigación indica que la asociación de una infección fúngica de los pulmones en un paciente con tuberculosis pulmonar aumenta y acelera la actividad del bacilo tuberculoso, ya directamente o reduciendo la vitalidad y resistencia del paciente, de tal manera que se observa un curso más desfavorable de la tuberculosis, caracterizada por recaídas y un progreso relativamente rápido. Se sugiere que en esta acción recíproca de dos enfermedades infecciosas coexistentes existe algún factor, alguna influencia prototáxica o simbiótica, que causa que una superimpuesta infección de los pulmones con hongos patógenos aumente los estragos de la tuberculosis pulmonar.

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Multiple Simultaneous Acute Putrid Lung Abscesses

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Putrid lung abscess is usually due to the aspiration and lodgement of infected particulate matter in the bronchial tree.

In virtually all cases, a solitary abscess occurs. It is a rarity for more than one abscess to develop simultaneously, either unilaterally or bilaterally. Theoretically, multiple abscesses are likely to be produced by marked flooding of the bronchial tree, such as in submersion.

Multiple hematogenous putrid pulmonary abscesses may ensue during septicemia, due to anaerobic organisms, but the fatal outcome of these patients is so rapid, their clinical course is quite different from that of the aspiratory type of abscess.

Ordinarily, the etiological factor in the development of a putrid lung abscess can be ascertained by eliciting a careful history. The mechanism producing such an abscess usually occurs during a general anaesthesia, unconsciousness due to any cause, or during operations upon the mouth, nose, or throat. That aspiration can take place during sleep has also been recognized clinically. Therefore, when a definite causative factor cannot be obtained, the presence of carious teeth, pyorrhoea, peridental pockets, or infected tonsillar crypts may be considered as the source of the aspirated material. The patient presented here had many carious teeth and a severe pyorrhoea with many pockets containing foul-smelling material.

Pulmonary infarction with later superimposed anaerobic infection may be the factor in the production of a putrid pulmonary abscess. However, when one considers the large number of pulmonary infarctions seen clinically and the very small number of these that develop into abscesses, it must be realized that a putrid lung abscess produced in this manner is indeed a rare occurrence.

During the course of an acute putrid lung abscess, one seldom finds either local or bronchogenic spread during the first six weeks. Therefore, when three separate lesions are found within ten days of the onset of the illness, one can assume that they have occurred simultaneously. Further corroborative evidence for

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FIGURE 1

FIGURE 2

FIGURE 3

Fig. 1: Hydropneumothorax at the right base, shadow in the right upper lobe, circular infiltration in the left upper lobe. *Fig. 2:* Empyema drained, right upper lobe shadow denser, left upper lobe infiltration still present.—*Fig. 3:* Fluid level in the right upper lobe, rarefaction in the left upper lobe.

this pathogenesis in the case reported here is that the patient had only a nonproductive cough during this period. This would virtually eliminate the possibility of a bronchial spread.

The presence of intermittent auricular fibrillation in this patient raises the question of the possibility of multiple pulmonary infarctions with later anaerobic infection. The x-ray findings, however, were in no way suggestive of infarctions; the patient had no pain until perforation into the pleura had occurred, and there was no hemoptysis. Furthermore, there had been no previous history suggestive of infarction, nor any later.

Because of the fact that this patient resided with his son-in-law, a physician, one can assume that the development of these putrid abscesses was asymptomatic. "Surprise putrid empyema" has been described by Neuhof and Stats.[1]

The different clinical courses of these three separate lesions ran the gamut of the clinical behavior of an acute putrid lung abscess. One healed spontaneously; another perforated, producing a putrid pyopneumothorax; and the third required surgical drainage.

CASE REPORT

A. D., No. 34223: A white male, 60 years of age, was admitted to the Private Pavilion of Montefiore Hospital on December 27, 1941. The history was obtained from his son-in-law, a physician, with whom he resided.

Past History: The patient was a mild hypertensive (170/90) for the past two years. He was known to have had a mild diabetes of many years' duration. He had never taken insulin. On a number of occasions he had had auricular fibrillation which usually responded rapidly to digitalis.

Present Illness: Six days before admission, the patient had a cough, with fever up to 100.5° F. Three days later, his heart began to fibrillate and this continued intermittently up to the time of admission. At the same time, he complained of pain in the right lower chest, and for the last 48 hours he had intractable hiccough.

Physical Examination: An elderly male, with persistent hiccough. The teeth were in very poor condition; there were many caries, considerable pyorrhoea, and pockets containing foul-smelling detritus.

Examination of the chest revealed dull percussion note over the right chest posteriorly. Blood pressure was 170/90. Urine showed 2 plus sugar, with no acetone. Hemoglobin was 16 gm. RBC, 5,900,000. WBC, 20,000. Polynuclears, 85 per cent. Blood sugar was 176 mgm. per cent.

X-ray examination performed on December 30, 1941 (Fig. 1) showed a hydropneumothorax at the right base posteriorly, a shadow in the anterior axillary part of the right upper lobe, and a small circular infiltration in the left upper lobe at the level of the anterior end of the second rib.

Aspiration of the right pleural cavity yielded thick foul-smelling pus. A diagnosis of putrid pyopneumothorax due to a ruptured putrid lung abscess was made. An operation was performed immediately.

Under local anaesthesia, liberal sections of the 8th and 9th ribs were removed. A bilocular empyema cavity containing considerable foul-smelling pus and fibrin was entered. It was situated directly upon the diaphragm, and the posterior loculation extended upward to the apex of the lower lobe, near the mediastinum. In this region, the ruptured pulmonary abscess was found. It was partly covered by a roof of necrotic pulmonary tissue. The empyema cavity was cleared of all debris and exudate, the roof of the lung abscess was excised widely, thoroughly exposing the interior. The pulmonary abscess and empyema cavity were tightly packed with gauze. Smear of the pus showed gram-positive bacilli and gram-positive cocci in long and short chains. Culture yielded streptococcus viridans.

Following operation, the patient became quite disoriented, but his general condition remained good. Glycosuria persisted without evidence of acidosis. The first dressing of the pulmonary-pleural cavity showed marked clearing of the infection, with no retention.

X-ray examination on January 9, 1942 (Fig. 2) showed clearing and diminution in size of the empyema cavity. The small infiltration in the left upper lobe was fainter and the shadow in the right upper lobe was denser and more localized. A subsequent examination on January 14th (Fig. 3) showed further improvement in the empyema cavity, an area of rarefaction in the left upper lobe infiltration, and a fluid level in the extreme anterior axillary part of the right upper lobe. This was so close to the thoracic wall that it suggested the presence of a perforation of another abscess with a pyopneumothorax.

The patient was taken to the operating room on January 14, 1942 and a segment of the right second rib in the anterior axillary line was removed. The pleura was found to be thickened, and aspirations yielded foul-smelling pus. A large putrid lung abscess was entered. There were a number of small loculations. The entire roof of the abscess was removed and the cavity packed.

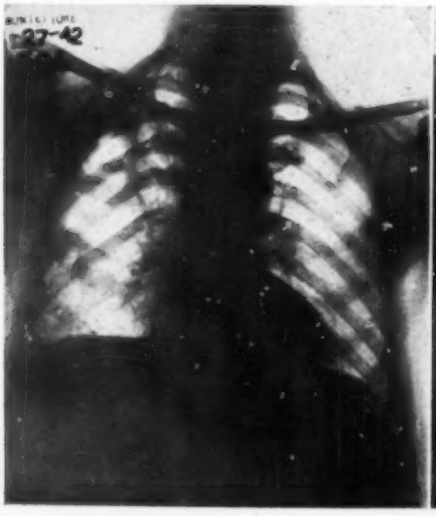

FIGURE 4

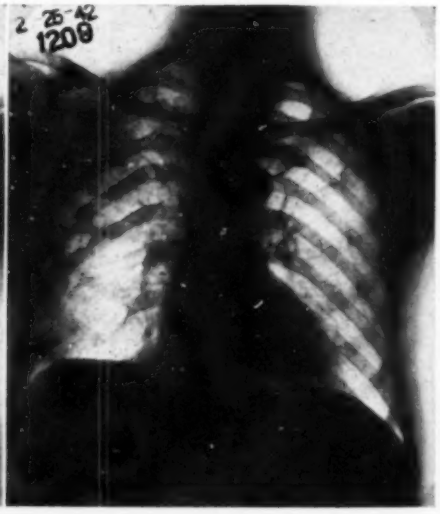

FIGURE 5

Fig. 4: Clearing of the empyema cavity and right upper lobe operative area, left upper lobe abscess smaller.—*Fig. 5:* Disappearance of left upper lobe lesion and continued clearing of the right hemithorax.

Following this operation, the general condition of the patient steadily improved; the mental confusion disappeared, and the urine became negative for sugar. Both wounds cleared up quickly and the bronchial fistulas, present in both, closed rapidly. An x-ray examination on January 27, 1942 (Fig. 4) showed continued improvement in the empyema cavity, a clearing of the right upper lobe operative area, and beginning disappearance of the left upper lobe abscess.

Both wounds healed rapidly and an x-ray examination on February 25, 1942 (Fig. 5) showed the disappearance of the left upper lobe infiltration and practically complete return to normal of the right hemithorax. The patient was discharged on February 26, 1942. Follow-up examination a number of months later showed normal pulmonary fields on fluoroscopic examination, and the patient had no symptoms referable to his respiratory system.

He remained in good health until 1946, when he was re-admitted to the hospital with a benign hypertrophy of the prostate. He died on August 6, 1946, following transurethral resection. No permission for post-mortem examination was obtained.

SUMMARY

Multiple simultaneous acute putrid lung abscesses are a rare clinical occurrence.

A case is reported in which three distinctly separate acute putrid lung abscesses appeared within a week of onset of symptoms.

The etiological factor was aspiration of infected particulate matter from the mouth.

Each abscess behaved differently—one perforated, causing a localized putrid pyopneumothorax which was drained; another disappeared spontaneously; and the third was treated by surgical drainage.

A complete cure was obtained.

RESUMEN

Los abscesos pulmonares pútridos agudos múltiples y simultáneos son de rara ocurrencia clínica.

Se informa sobre un caso en el que tres abscesos pulmonares pútridos agudos, completamente separados, aparecieron en la semana de la iniciación de los síntomas.

El factor etiológico fue la aspiración de materia infectada proveniente de la boca.

Cada uno de los abscesos reaccionó diferentemente: uno se perforó y causó un pioneumotórax pútrido localizado que fue canalizado; otro desapareción espontáneamente y el tercero fue tratado por canalización quirúrgica.

Se logró la completa curación.

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Tuberculosis in Pediatric Practice

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Optimism over the complete eradication of tuberculosis as a major cause of illness and death in the United States seems justified. Some estimate that this goal may be reached in as short a time as a generation or two. Should a drug such as streptomycin, in the meantime prove out to be effective against the disease, the time would of course be shortened.

A glance at recent statistics gives the basis for such optimism. In 1944 the tuberculosis mortality rate was 41.3 for all forms of tuberculosis, and if the pulmonary type alone is considered the rate was only 38.3. Contrast this with the rate of 201.9 deaths per 100,000 population for the U. S. Registration Area in 1900. Tuberculosis now occupies seventh place in the list of causes of death instead of first place where it stood for so many years. While it was once held that tuberculous infection after the tenth year of life was practically universal it is now probable that considerably less than one-half of the adult population reacts positively to tuberculin, and the proportion of young persons reaching adult life with negative tests is steadily growing. The Committee on Tuberculosis of the American Student Health Association reported for the year 1942-43 that among 208 colleges and universities representing a total student enrollment of 300,144, the incidence of positive reactors was 18.6 per cent. In some rural schools in the Middle West as few as six to ten per cent of the children react positively to the tuberculin test. Indeed, in one four-county sanatorium district in Minnesota in 1946, out of 240 schools surveyed not a single reactor was found in 183 of them. When a similar result has been achieved for a majority of the counties of the nation the conquest of human tuberculosis will have become as successful as has the conquest of bovine tuberculosis by the veternarians.

But in spite of these encouraging indications a monumental task yet remains to be accomplished. Great progress was made during the war when millions of young men were examined for tuberculosis by roentgenograms of the chest. This important work is being carried on throughout the country among the civilian population by means of mobile x-ray units using micro-film. The objective of course, is to locate persons harboring tuberculous disease so that they may be isolated and brought under treatment

at the earliest possible moment. The purpose of this article is to stress the contribution physicians, in their every day practices among children, can make in this final concerted drive against one of the worst scourges of mankind. In the following paragraphs diagnostic tuberculin, pathogenesis of the common forms of tuberculosis encountered in children, and their diagnosis, prognosis, prevention and treatment are discussed.

In diagnostic tuberculin the physician has at his disposal one of the most specific of all the specific tests available for the detection of disease. It should be used as routinely in one's daily practice to exclude tuberculosis as is the Wassermann or a similar test to exclude syphilis, or tests of the urine for sugar and albumin to exclude diabetes and nephritis, or the agglutination tests for typhoid or malta fever. Negative tests may be as significant as positive tests. Recently a ten year old girl was under observation because of a lesion of her lower thoracic spine of many months duration which had resulted in a kyphosis. X-ray films were interpreted by the roentgenologist and orthopedist as tuberculous spondylitis. However, her tuberculin test, even with five milligrams of O.T., was consistantly negative. Operation became necessary because of pressure on her spinal cord. Examination of material removed proved the lesion to be due to an osteogenic sarcoma. Repeated experiences such as this over a good many years have led me to be extremely reluctant to accept any lesion as tuberculous, however suspicious, when properly performed tuberculin tests with reliable materials are negative.

True, there are a few exceptions in which tuberculin tests may react negatively even when a tuberculous infection exists. These may be enumerated as follows: first, in the pre-allergic period, that is, after infection occurs but before allergy develops; second, during certain of the acute communicable diseases, such as whooping cough, measles, scarlet fever, and diphtheria; third, when the tuberculous lesions have become completely healed (no viable bacilli) so that no tuberculoprotein reaches the circulation; and fourth, when overwhelming tuberculous disease depresses tissue reactibility to a state of anergy. With these few exceptions the tuberculin test may be accepted as providing a very efficient method for sifting out those who have not been so infected. Furthermore, a negative test in a child offers the strong probability that the members of his family with whom he is in close contact are also free from tuberculous disease, at least in a communicable form.

In evaluating the meaning of a positive reaction to tuberculin, one must remember that sensitivity of all the tissues of the body results within a few weeks following the establishment of the

primary infection. Since allergy thus created continues in the majority of individuals throughout life, it follows that a positive reaction at any age denotes only that a primary infection has taken place. Its diagnostic implications, therefore, extend no further than this. For location, extent, activity and stage in the evolutionary course of the disease, other methods of study must be undertaken. Furthermore, no significant correlation exists between the size of the reacting area and the amount of activity of the tuberculous disease present.

However, a positive reaction to tuberculin is significant in several other respects. In the first place, it means that the child has at some time been in contact either with a human or bovine source of infection. Herein lies one of the most important contributions the pediatrician or the general practitioner can make in the conquest of tuberculosis. By doing routine tuberculin tests on his child patients, the physician may be able to uncover an hitherto unsuspected source of contagion. The younger the child who shows up with a positive test the more likely it is that the source of the tubercle bacilli is someone in the immediate family. Old persons with chronic coughs should particularly come under suspicion; but the job is not done until every member of the family as well as other possible sources have been investigated for tuberculosis. The grave importance of locating the spreader of tubercle bacilli is too well known to need further comment. But that the child with a positive tuberculin test can frequently lead to the detection of an open case of tuberculosis is a fact not taken advantage of as frequently as it should be. Finally a positive test in a child identifies the individual who must be kept under observation for the possible development of chronic pulmonary tuberculosis at some future date. This form of tuberculosis rarely makes its appearance before the age of puberty, but knowledge of pre-existing sensitivity to tuberculin at or before this time singles out those who should have yearly chest roentgenograms, not only during the teen age period when the incidence of pulmonary disease is high, particularly in girls, but for years afterwards. It is conservatively estimated that one out of every ten children with a positive reaction sooner or later develops chronic pulmonary tuberculosis. Here again the physician in private practice has a real responsibility for detecting this form of tuberculosis at its very beginning, before the patient has become a spreader of organisms and while treatment is promising and relatively simple. Such early recognition is possible only by roentgenography. Early diagnosis based upon symptoms and physical findings invariably means for advanced tuberculosis. A passing thought which might be recorded here is that if the present vogue of x-raying chests of children

with miniature films leads to a decrease in the use of diagnostic tuberculin much information of value in the control of tuberculosis will be lost.

Pathogenetic Development of Tuberculosis

Tuberculosis is essentially a biphasic disease consisting of primary and reinfection phases. The primary phase most frequently occurs in childhood, and formerly was referred to as "childhood type of tuberculosis." However, it also occurs in adults and with increasing frequency in recent years for the simple reason that fewer children are being infected. The term reinfection phase has supplanted the older term of adult type of tuberculosis, largely for the reason that while it most commonly occurs in adults it may also occur in children. There are certain fundamental differences between these two phases.

Primary Tuberculosis	Reinfection Tuberculosis
1. Organism seeded on nonallergic tissues.	1. Organisms seeded on allergic tissues.
2. Progression occurs readily via lymphatics to regional lymph nodes.	2. Little tendency for involvement of regional lymph nodes.
3. Spread by lymphohematogenous route characteristic.	3. Chief method of spread is by contiguity and bronchogenic.
4. Marked tendency for lesions to recede and calcify.	4. Tendency for lesions to progress, to cavitate, and to spread.
5. As a rule evidences of illness are slight.	5. Accounts for practically all of major morbidity and mortality from tuberculosis.

The precise definition of these two phases is still a matter of controversey. Myers is the chief exponent of the view which restricts the definition of the primary phase to include only the lesions set up as the direct result of the initial infection in the period before allergy becomes established. Any new set of tuberculous lesions occurring subsequent to the development of allergy and outside of the original lesions is considered as belonging to the reinfection phase. Diagnostic Standards for 1940, on the other hand, defines the primary phase as including all forms of tuberculosis which follow directly and uninterruptedly the first implantation of tubercle bacilli. Thus, tuberculous pneumonia, miliary tuberculosis, and tuberculosis meningitis are classified as belonging to the primary phase, whereas in Myers' conception these are classified as acute reinfection forms. The essential difference in these points of view would seem to be largely in their prognostic implication. Myers considers primary tuberculosis as a routinely benign disease, but it cannot be regarded as such, particularly in young children, if one accepts the definition advanced by Diagnostic Standards.

While primary infections may occur in the abdomen, in the cervical region, or in the skin, by far the commonest route (over 90 per cent) is by way of the respiratory tract. The first effect of inhalation of virulent tubercle bacilli is the establishment of single or multiple foci of tuberculous pneumonia in some part of the lung, usually close beneath the pleura. Bacilli multiply here and are carried by the lymphatics to one or more of the regional broncho-pulmonary lymph nodes. The pulmonary focus and lymph node focus together constitute the "primary complex" of Ranke.

Allergy becomes established within two to seven weeks from the time of the initial infection. Subsequently the lesions of the primary complex usually undergo a process of gradual retrogression, encapsulation, calcification, and occasionally ossification. Viable tubercle bacilli may remain locked up in these calcified foci for months, years or even the life time of the individual. The complete developmental course of the primary phase most frequently runs its entire course without the knowledge of the patient, family or physician. In approximately three-fourths of children who have undergone this experience the only residual evidence is a positive tuberculin reaction. Of the remaining twenty-five per cent the majority will show in the x-ray film characteristic calcified foci either in the lung parenchyma, hilus nodes, or both.

Occasionally, and most often in young children and those known to have been exposed, the lesions of the primary infection may be detected by the x-ray in the early or fresh state. In such cases symptoms and slight physical findings may be present. The most important symptom appearing at this time is fever. It usually begins coincidentally with the development of allergy and continues for a week, a month or even longer. However, there is little to distinguish this fever from that occurring in ordinary acute infections so that its significance is frequently not realized unless a positive tuberculin reaction raises the possibility. Temporarily the fever may be accompanied by lassitude, anorexia and failure to gain weight, but on the whole the evidences of illness are slight. Physical findings are usually meager or absent altogether, even when rather prominent pneumonic shadows are revealed in the x-ray film. Occasionally, however, a few rales or changes in breath sounds may be elicited over the involved area.

The cause of these pneumonic shadows has been the subject of some debate. Eliasberg and Neuland in 1920 employed the term "epituberculosis" to describe them in the belief that although they occurred in tuberculin sensitive children, they were of a non-tuberculous nature. More recently the suggestions have been advanced that they represent atelectatic areas due to obstruction of a bronchus from lymph node pressure or to obstruction from

allergic swelling of the mucous membrane inside the bronchus. The prevailing view, however, favors the belief that the cause of these shadows is an allergic inflammatory reaction about a tuberculous nodule and its surrounding zone of tubercles. The further course of these pneumonic infiltrations is usually one of slow retrogression over weeks and months, the densities often persisting long after symptoms and physical findings have subsided. Central caseation probably occurs in most of them, but rarely are cavities demonstrated by x-ray. The eventual outcome is one of slow resolution, either to complete disappearance of the lesions, to a few fibrous strands along the course of the lymphatic channels leading to the lymph node component, or to calcified deposits (Ghon tubercles) in the caseous centers.

While hilus gland tuberculosis is invariably associated with the pulmonary component of the primary complex, at times it may appear to be present alone. The tuberculous lesions exist in the nodes as caseous, partly caseous and calcified, or calcified. Occasionally, the caseous stage is considerably prolonged, but as a general rule, encapsulation, resolution and calcification more or less parallel similar changes in the pulmonary focus. In the majority of instances, the caseous nodes are not sufficiently enlarged to be visualized in the x-ray film. Symptoms are either absent or of such a mild general character that no specific significance can be attached to them, and even if the tuberculin reaction is positive, a definite diagnosis cannot be reached until serial x-ray studies reveal the beginning deposition of calcium. With a further increase in severity, some of the nodes may protrude beyond the margins of the mediastinum and become demonstrable in the x-ray film as characteristic rounded knobs. In this stage, fever, anorexia, anemia, hoarseness, and a bitonal cough may or may not be present.

Rarely, and again usually in the early years of childhood, massive enlargement of the mediastinal nodes literally chokes the whole area in the vicinity of the root of the lung resulting in compression of the bronchi, trachea, esophagus, blood vessels or nerves. Under such circumstances, severe and alarming symptoms may develop. The usual course of hilus gland tuberculosis, however, is one of healing by resolution and calcification over a period of from six months to two or three years.

Again, and most often in infants and young children where the opportunities for massive doses of bacilli are greatest, the lesions of the primary complex may not promptly retrogress, but on the other hand may undergo progression and extension. Thus, progress may occur at the site of the primary pulmonary infiltration by contiguity, leading sometimes to a large excavating pneumonia. Severe toxic symptoms may develop in which cough, cyanosis,

irregular and persistent fever, loss of weight, anemia, prostration and rapid pulse predominate. Hemoptysis may occur. Physical findings of extensive dullness, tubular breathing and rales are readily obtainable over the involved area. Sputum obtained by gagging or stomach washings contains tubercle bacilli in large numbers. For many weeks or months, there may be grave doubt as to whether one is dealing with a lesion which will eventually resolve and calcify or one which will progress and result in death.

Ulceration of a cavity or a caseous lymph node into a bronchus with aspiration of large numbers of tubercle bacilli into the lung results in acute tuberculous pneumonia.

Tuberculous pneumonia, unlike primary pulmonary infiltrations, tends to progress, to spread, to cavitate and to be attended by grave symptoms of illness. At first, the disease may be unilateral, but as a rule the other lung before long becomes involved through transbronchial spread. The onset of the symptoms may be acute, simulating non-tuberculous pneumonia, or it may be more gradual. In either case, the picture shortly is one of severe prostration with fever, cough, dyspnoea, cyanosis, and rapid failure predominating. Hemorrhage may occur as the result of rupture of a blood vessel into a cavity. Physical findings are usually positive, resembling those of ordinary pneumonias with the possible exception that they may not be as extensive as one would expect from the roentgenographic picture. The disease is to be differentiated chiefly from the acute pneumonias, and from large primary pulmonary infiltrations. In respect to the latter, one may be in doubt for sometime, but the x-ray evidence of spreading lesions, particularly when they appear in the opposite lung, usually serves to make the distinction. Collapse therapy may be indicated in this form of tuberculosis.

Progress from the primary lesions by way of the lympho-hematogenous route is of considerable significance. During the pre and post-allergic period tubercle bacilli may reach the general circulation in dosages of varying degrees. Transported by the blood the bacilli may cause single or multiple lesions in the lungs, meninges, bones, kidneys, skin and other organs. If the dosage is not too great, such foci usually become encapsulated and remain quiescent, but always as potential sources of clinical disease at some future time. When overwhelming invasion of the circulation occurs, as from rupture of a caseous node directly into a large blood vessel, the result is miliary tuberculosis, generalized or pulmonary, a complication few children survive. Until recently the pathogenesis of tuberculous meningitis was generally regarded as being similar to that of miliary tuberculosis in that tubercle bacilli were carried directly to the meninges via the blood stream

from a focus in the tracheobronchial lymph nodes. Rich and McCordock, however, have presented convincing evidence that this is not the case. Miliary tuberculosis apparently sets up caseous foci in the substance of the brain, cord, meninges, choroid plexus or in the bones encasing the central nervous system. It is the rupture of one or more of these older caseous foci, permitting the discharge of bacilli directly into the cerebrospinal fluid which originates diffuse tuberculous meningitis. This view is supported by the fact that generalized miliary tuberculosis and diffuse tuberculosis meningitis occur independently. Furthermore, the experimental injection of large numbers of virulent tubercle bacilli into the carotid artery produces occasional sparsely located meningeal tubercles rather than diffuse tuberculous meningitis.

It is probable that from ten to twenty-five per cent of all children who acquire a primary tuberculous infection subsequently develop reinfection tuberculosis of the chronic pulmonary type. Only rarely does it develop in children before the tenth year of life, but it appears with increasing frequency during adolescence and reaches its maximum incidence between eighteen and thirty to thirty-five years.

The time interval elapsing between the initial phase of the primary infection and the first development of the reinfection pulmonary disease may be many months or years, or the interval may be so brief that one appears to be practically continuous with the other. The former is likely to be the situation when the primary infection occurs sometime during childhood, but before puberty, while the latter more often obtains when the primary infection takes place in adolescence or in young adult life. No certain explanation exists for the unusual freedom which children between the ages of infancy and adolescence seem to enjoy from phthisical forms of tuberculosis. It has been suggested that it is due to the long period of time necessary for tuberculosis to develop into clinical disease in the human body, and that a similar course occurs in the young of animals and fowl. Also, it is likely that the increased need for minerals and proteins and other metabolic changes which occur in adolescence in association with the establishment of menses as reported by Johnson have a significant bearing upon the development of tuberculosis in puberty and upon its more frequent occurrence in adolescent girls.

The tubercle bacilli responsible for pulmonary tuberculosis may originate from exogenous or endogenous sources. Possible endogenous sources are the lesions of the primary complex from which bacilli may escape, or a pulmonary lesion laid down during the pre or post-allergic lympho-hematogenous spread may light up. Whatever the source, the earliest lesions consist of small areas

irregular and persistent fever, loss of weight, anemia, prostration and rapid pulse predominate. Hemoptysis may occur. Physical findings of extensive dullness, tubular breathing and rales are readily obtainable over the involved area. Sputum obtained by gagging or stomach washings contains tubercle bacilli in large numbers. For many weeks or months, there may be grave doubt as to whether one is dealing with a lesion which will eventually resolve and calcify or one which will progress and result in death.

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of lobular pneumonia usually from one to three centimeters in diameter, which in the x-ray film appear as soft mottled shadows most frequently located in the subapical region. Usually a single focus, most often in the right lung, occurs, but multiple foci may be present. The ultimate fate of these early infiltrates may be in doubt for several months or even years. Some retrogress and heal by fibrous encapsulation, resorption or occasionally by calcification. Others undergo a slow process of progression by contiguity, central caseation, liquifaction and cavity formation.

Diagnosis of chronic pulmonary tuberculosis in its earliest incipient stages can be made only by x-ray. It is important, therefore, that tuberculin sensitive children should have annual x-ray films made of the lungs from the age of puberty on. The detection of the characteristic soft mottled apical shadow of the early infiltrate calls for a most careful period of observation. Once the tuberculous nature of the lesion has become established and evidence of progression obtained specific treatment is indicated. In the majority of cases this will take the form of some type of collapse therapy.

Diagnosis

Diagnostic tuberculin and the x-ray constitute the bulwarks upon which chief dependence is placed for the diagnosis of the various forms of tuberculous disease occurring in childhood.

History of exposure, symptoms of illness and physical findings are relatively poor diagnostic guide posts. Among those who are known to have been exposed to an open case of tuberculosis it may be expected that the proportion found to be infected will be much greater than where no such history is obtained. On the other hand, active tuberculosis in a communicable stage is not infrequently present in some adult member of the family, or close associate of the child, entirely unsuspected. Unfortunately, the symptomatology of tuberculosis is not pathognomonic with the possible exception of the advanced stages of some of the reinfection forms. The transitory fever which accompanies the acute phase of some primary infections is easily overlooked, or if it is noted, is ascribed to nonspecific infections of the respiratory tract. A similar situation exists with respect to physical findings.

Nevertheless, a careful physical examination is not to be neglected since it may be of great importance in the differential consideration of nontuberculous disease. In this respect, one should remember that positive physical findings obtained over the lungs, such as rales and changes in breath sounds in the percussion note, are in the vast majority of instances due to conditions other than tuberculosis. Underweight, poorly nourished children are no more susceptible to tuberculous infections than are their robust asso-

ciates. While children who are known to have been exposed to open cases of tuberculosis, and those who present symptoms or physical signs of illness, form an obvious group in which investigation for tuberculosis is clearly indicated, nevertheless, restriction of case finding, even in private practice, to such children will inevitably result in failure to discover the major portion of infected children. The routine use of diagnostic tuberculin for children of all ages, with retesting of the negative reactors at least every three years is a practicable program.

For determining the location and extent, and for following the course of tuberculous lesions, the roentgenogram or fluoroscope is indispensable. In general, the child who is discovered to react positively to tuberculin should have at least one x-ray film of his chest at that time. Experience has shown that further x-ray study may, in the vast majority of instances, safely be postponed until the tenth year of life.

Demonstration of tubercle bacilli is desirable whenever possible since it clinches a previously suspected diagnosis. Because of the habit most young children have of swallowing their sputum, the usual method of sputum examination as employed in older children and in adults is successful only occasionally when sputum can be obtained by gagging on a swab or on the end of a tongue blade. However, it has been shown that tubercle bacilli may be recovered from the stomach contents or in the feces of a fair proportion of children with primary intrathoracic tuberculosis. As might be expected, positive results are most frequently obtained in the younger patients. Within six to eight months after the initial primary infection, there is a marked tendency for the stomach contents to become negative, indicating that this is the length of time required for closing of the primary foci. What public health liability there is from children with primary infections would seem to be largely confined to this relatively brief period during which the lesions are in a "fresh" stage.

The technic of obtaining gastric contents for examination consists of washing the stomach with two to three hundred cubic centimeters of sterile water in the morning after a fast of six hours. The material is centrifuged, the sediment homogenized and a stained smear is examined. If negative, cultures are made and animal inoculation carried out. Similarly, material obtained from other suspected sources such as pleural effusion, ascitic fluid, cerebrospinal fluid and cold abscesses should be examined.

Studies of the blood, including the hemogram, sedimentation rate and Schilling differential count, are of less value in the diagnosis of tuberculous infections in children than in estimating prognosis and the favorable or unfavorable course of the disease.

It is important to keep in mind the variations which normally occur in different age periods between lymphocytes, monocytes and leukocytes. No significant changes are noted in the total leukocyte count or in the differential smear in any of the stages of primary tuberculosis, including the acute inflammatory stage. In cases of reinfection tuberculosis an increase in the total leukocyte count is suggestive of secondary infection; a relative increase of monocytes over lymphocytes (M.L. ratio) is indicative of activity and progression of the tuberculous disease; the reverse of these relationships occurs with resolution and healing; and a shift to the left in the Schilling count is evidence of increased toxicity, the greater the shift the more serious the prognosis. Likewise repeated determinations of the sedimentation rate of the red cells may be useful in estimating the activity of the tuberculous process and in following its course.

Prognosis

The immediate prognosis of all primary tuberculous infections is good with the exception of infections occurring during the first five years of life, particularly during the first two years. In this age period the prognosis is considerably worse for colored children than for white children. Approximately ninety per cent of white children infected during the first five years of life may be expected to survive their tuberculous infection. Such mortality as there is from tuberculosis will occur largely in the first two years of life and will occur in the group whose chest roentgenograms show parenchymal lesions. The risk to life is nearly twice as great in infants who acquire their tuberculous infection in the first year of life as in those who do not become infected until the second year. Death is due almost exclusively to the acute reinfection forms of tuberculosis such as meningitis, miliary disease and tuberculous pneumonia which result from the extension of the disease beyond the confines of the primary complex.

The remote prognosis for children who have acquired primary infections during childhood is uncertain. An unknown number (perhaps from ten to twenty per cent of those who enter puberty with positive tuberculin reactions) will fall ill with chronic reinfection pulmonary tuberculosis sometime during adolescence or early adult life. Of this number at least one-fourth will die of their tuberculous disease within the next six years. Whether institution of collapse therapy in the pre-symptomatic minimal stage will greatly alter this poor prognostic outlook in the future remains to be seen. It is very probable that it will.

Prevention

The hope of complete eradication of tuberculosis from the human race seems to rest squarely on the basis of its control by epidemiologic means. Already great progress has been made in this direction. Bovine infections have been practically eliminated as a source of danger to infants and children. Protection of children from human infections is, of course, a far more difficult task, but the means are at hand awaiting application only to accomplish it. If a leaf could be borrowed from the methods employed by the veterinarians, the entire population of the nation would periodically be tested by tuberculin, the positive reactors would be x-rayed and studied for clinical disease, and those found to be contagious would be isolated and treated. While such an ideal program has little chance of immediate attainment, it can and is being approached. It is entirely practicable for physicians to provide their child patients with an immediate environment which is safe from the risks of tuberculous infection. A chest roentgenogram and tuberculin test should be as routine in the prenatal care of every woman as is the taking of a blood specimen for syphilis. A similar examination should be made of the members of the family including domestic workers, of school teachers, and of physicians, nurses and other personnel of hospitals caring for children. Repetition of these precautionary measures every two or three years, together with testing of the children themselves with tuberculin at like intervals, constitutes the most effective prophylactic program for children available at the present time.

Further protection for the children of America is being secured by mass x-raying surveys of apparently healthy groups in industry, rural areas, high schools, colleges and other groups. These programs are usually carried on without preliminary tuberculin testing and have as their aim the detection of active or potentially active tuberculous lesions. Miniature films and rapid film taking methods make it possible to examine large numbers of persons quickly and economically.

Treatment

Unfortunately, no specific form of drugs or serum therapy has been developed for tuberculosis. The sulfonamide chemicals apparently are not effective against the tubercle bacillus. Streptomycin is still in the experimental stage. However, it offers the most promise of any of the newer chemicals so far brought forward.

Rest, diet, healthful living conditions, and time are the general measures to be depended upon for bringing about healing in most of the tuberculous lesions. The importance of breaking contact with sources of infection as the first step in any form of tuber-

culous disease in a child cannot be overemphasized. The great majority of primary infections need no further treatment than this, plus rest and a well balanced diet for a few months when the lesions are discovered in the acute stage. Sanatorium or preventorium care is unnecessary, although the educational value of such institutions cannot be denied. Their facilities and those of convalescent or foster homes should, however, be utilized when contact with infection cannot be broken in the child's own home. Every precaution should be taken, too, to avoid exposing children with active tuberculous lesions to the acute communicable diseases, including colds and influenza. Ascorbic acid requirements are considerably increased in tuberculosis; hence liberal amounts of this vitamin should be included in the diet. Resumption of normal activities should be gradual, and presaged upon the serial x-ray appearance of lesions, the absence of symptoms, on the sedimentation rate and upon an optimum state of nutrition.

Heliotherapy is indicated in all forms of tuberculosis except active intrathoracic lesions. Exposures should be gradual and should be interdicted if an increase in fever and pulse rate is noted. Sunlight is preferable to artificial sources of ultraviolet rays. Cold or fresh air on open porches has been abandoned in favor of controlled ventilation and moisture content. The field of usefulness of collapse therapy in children is almost entirely confined to the treatment of chronic reinfection pulmonary tuberculosis in puberty and adolescence. However, it may have to be considered occasionally in younger children when cavities or hemorrhages occur in connection with the lesions of massive primary pulmonary tuberculosis or tuberculous pneumonia.

SUMMARY

Complete eradication of tuberculosis as a major cause of illness and death in the United States within the next generation or two is an attainable possibility by application of methods already known. If streptomycin or a similar drug proves efficacious this time would be considerably shortened. In the over-all fight against the disease the physician whose practice deals with children can contribute much in this final drive.

Diagnostic tuberculin is one of the most specific of all the specific tests available for the detection of disease. The physician should use it routinely in his child patients at two to three year intervals. False negative tests occur only rarely. A positive test means only that a primary tuberculous infection has occurred at some previous time. However, the finding of a positive reaction in a child, especially a young child in the first years of life, may be the means of uncovering an hitherto unsuspected source of con-

tagion. Furthermore, a positive test in a child identifies the individual who must be kept under observation for the possible development of chronic pulmonary tuberculosis at some future date. Mass x-raying the chests of children fails to make this distinction.

Knowledge of the pathogenetic development of the various forms of tuberculosis aids the physician in recognizing the stage in the evolutionary course of the disease at which his patient may have arrived. There are fundamental differences between primary tuberculosis and reinfection tuberculosis. The development of tuberculo-allergy several weeks after the initial infection is responsible for these differences. In the majority of children primary tuberculosis is limited to the pneumonic lesion and to the lymph node lesion. In a few, extension may occur by contiguity about the pulmonary focus, or by hematogenous or bronchogenic spread from the lymph-node focus. Thus accounted for are the acute reinfection forms of tuberculosis such as tuberculous broncho-pneumonia, miliary tuberculosis, and tuberculous meningitis. The latter, however, is not the direct result of hematogenous seeding, but rather, results from rupture of an older focus into the sub-arachnoid space.

Chronic pulmonary tuberculosis is rarely encountered before puberty. No certain explanation exists for the remarkable freedom children between the ages of five and puberty enjoy from tuberculous morbidity and mortality. Possible factors having a bearing upon the high infection rate of chronic reinfection tuberculosis, especially in girls, during the teen age are the increased need for minerals and protein and other metabolic changes which occur in adolescence in association with the establishment of menses. The reaction of every child to tuberculin should be known at or before the onset of puberty. Positive reactors should have, at least annual x-ray films of the lungs made in order to recognize at the earliest possible time, the characteristic soft mottled apical shadows of the early infilliate.

Diagnostic tuberculin and the x-ray constitute the bulwarks upon which chief dependence is placed for the diagnosis of the various forms of tuberculous disease occuring in childhood. History of exposure, symptoms of illness and physical findings are relatively poor guide posts. Demonstration of tubercle bacilli by gastric lavage is frequently helpful in clinching a previously suspected diagnosis. Studies of the blood, including the hemogram, sedimentation rate and Schilling differential count are of less value in diagnosis than in estimating prognosis and the favorable or unfavorable course of the disease.

The immediate prognosis of all primary tuberculous infections is good, with the exception of the comparatively few children in the first five years of life whose primary complexes extend to the

development of such acute reinfection forms as meningitis, miliary disease and tuberculous pneumonia. The remote prognosis however, is more uncertain, both from the point of view of the number who will ultimately develop chronic pulmonary tuberculosis and the number who will die from this form of the disease.

An environment safe from the risks of tuberculosis infection should be provided every child. In this preventive phase the pediatrician and the physician can accomplish much.

Experiments now being carried on with streptomycin may provide a specific form of therapy, but until its efficacy has been proven, rest, diet, healthful living conditions and time are the general measures to be relied upon for bringing about healing in most of the tuberculous lesions of children. Collapse therapy may occasionally be indicated.

RESUMEN

La completa erradicación de la tuberculosis como causa mayor de enfermedad y de muerte en los Estados Unidos dentro de la próxima generación o la siguiente, es una posibilidad realizable mediante la aplicación de métodos ya conocidos. Si la estreptomicina u otra droga semejante resulta eficaz, podría ser considerablemente acortado este plazo. En la lucha total contra la enfermedad, el médico que trata a los niños puede contribuir mucho en esta campaña final.

La tuberculina diagnóstica es una de las más específicas de las pruebas específicas disponibles para el descubrimiento de enfermedades. El médico debe usarla sistemáticamente en sus niños enfermos, a intervalos de dos o tres años. Las reacciones negativas falsas ocurren con rareza. Una reacción positiva sólo significa que ha ocurrido una infección tuberculosa primaria en una época previa. Sin embargo, el descubrimiento de una reacción positiva en un niño, especialmente en un niño pequeño, en los primeros años de vida, puede ser el medio de revelar una fuente de contagio hasta entonces no sospechada. Además una prueba positiva en un niño señala al individuo que debe ser observado por el posible desarrollo de tuberculosis pulmonar crónica en una fecha futura. Los exámenes radiográficos colectivos de los pechos de los niños no establece esta distinción.

El conocimiento del desarrollo patógeno de las varias formas de la tuberculosis ayuda al médico a reconocer la etapa en el curso evolutivo de la enfermedad en que el paciente puede encontrarse. Existen diferencias fundamentales entre la tuberculosis primaria y la tuberculosis de reinfección. El desarrollo de la alergia a la tuberculina que ocurre varias semanas después de la infección inicial, es responsable de estas diferencias. En la mayoría de los

niños la tuberculosis primaria está limitada a la lesión neumónica y a la de los ganglios linfáticos. En unos cuantos puede ocurrir una propagación por contigüidad alrededor del foco pulmonar, o una propagación hematógena o broncógena proveniente del foco en el ganglio linfático. Esto explica las formas agudas de la tuberculosis de reinfección, tales como la bronconeumonía tuberculosa, la tuberculosis miliar y la meningitis tuberculosa. Esta última forma, sin embargo, no es el resultado directo de una siembra hematógena, sino que se debe más bien a la ruptura en el espacio subaracnoideo de un foco más antiguo.

Sólo raramente se encuentra la tuberculosis pulmonar crónica antes de la pubertad. No existe una buena explicación de la razón por la cual los niños de los cinco años a la pubertad disfrutan de una extraordinaria inmunidad a la morbicidad y mortalidad tuberculosas. Los posibles factores que tienen que ver con el elevado coeficiente de infección de la tuberculosis de reinfección crónica, especialmente en las niñas de 13 a 19 años, son: el aumento en la demanda por minerales y proteínas y otros cambios metabólicos que ocurren en la adolescencia, asociados con el establecimiento de la menstruación. Debe conocerse la reacción a la tuberculina de todo niño durante o antes del comienzo de la pubertad. A los reactores positivos se les deben tomar películas radiográficas de los pulmones por lo menos anualmente, a fin de descubrir lo más temprano posible las suaves y moteadas sombras en el ápice que caracterizan el infiltrado precoz.

La tuberculina diagnóstica y la radiografía constituyen los baluartes en los cuales se debe poner la mayor confianza para el diagnóstico de las varias formas de tuberculosis que ocurren en la niñez. La historia de exposición, los síntomas de enfermedad y los hallazgos físicos, son postes indicadores de relativamente poco valor. La demostración de bacilos tuberculosos por medio del lavado gástrico a menudo ayuda a verificar el diagnóstico previamente sospechado. Los estudios de la sangre, inclusive del hemograma, índice de sedimentación y enumeración diferencial de Schilling, tienen menor valor en el diagnóstico que en calcular el pronóstico y el curso favorable o desfavorable de la enfermedad.

El pronóstico inmediato de todas las infecciones tuberculosas primarias es bueno, con la excepción del comparativamente pequeño número de niños en los primeros cinco años de vida cuyos complejos primarios progresan hasta el desarrollo de tales formas agudas de reinfección, como la meningitis, la enfermedad miliar y la neumonía tuberculosa. El pronóstico remoto, sin embargo, es más incierto, tanto desde el punto de vista del número de los que finalmente contraerán tuberculosis pulmonar crónica como del número de los que morirán de esta forma de la enfermedad.

Todo niño debe estar situado en un ambiente en el que se encuentre amparado contra los riesgos de la infección tuberculosa. En esta fase preventiva tanto el pediátrico como el médico pueden lograr mucho.

Los experimentos que actualmente se están llevando a cabo con la estreptomicina podrán suministrar una terapia específica, pero hasta cuando se haya comprobado su eficacia, el reposo, la dieta, las condiciones de vida saludables y el tiempo, son las medidas generales de las que se debe depender para promover la curación de la mayor parte de las lesiones tuberculosas en los niños. Ocasionalmente se indica la colapsoterapia.

Silicosis as an Industrial and Compensation Problem in British Columbia*

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Silicosis is an ancient industrial disease long known by various names, e.g., "grinder's rot," "potter's asthma," "miner's consumption," all of which indicate its industrial origin and rather hopeless prognosis. During the past twenty-five years, due to research work done by numerous scientists in various countries, and the greatly extended use of the x-ray, our knowledge concerning this disease has been widely extended. It still presents, however, many problems in its pathology, diagnosis and treatment, which are unsolved.

In this paper it is proposed to give, in a more or less dogmatic way, a summary of our present knowledge and a brief account of the methods used in British Columbia in prevention, treatment and compensation of its victims.

Etiology:

Silicosis is due to inhalation of fine particles of silica (SiO_2). The hazard of inhalation of silica dust is governed by the following factors:

1. The fineness of the particles. Particles must be five microns or less in size to be dangerous.
2. The concentration of the dust in the atmosphere and the per cent content of silica. Air that contains more than 300 particles per c.c. of dust, and contains 6 per cent or more of pure silica is considered hazardous.
3. Adulterating dusts inhaled with the silica. It has been known for some time that various dusts inhaled with silica not only acted as diluents but also as directly inhibiting the development of the disease.
4. A fourth factor is susceptibility of the workman. It is known that manifest tuberculosis increases the susceptibility of the individual to silicosis. Hence the importance of excluding workmen with known tuberculosis from exposure to silica dust. There are other unknown factors; e.g., other respiratory infections and unknown constitutional factors that increase

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susceptibility. It is hoped that future research will clear our knowledge on these important points.

Pathology:

Silica dust, when inhaled into lungs, is not inert like most dusts; e.g., coal dust. When it comes in contact with the body juices, silica becomes definitely toxic by the liberation of an irritant biochemical product. The phagocytes carrying the particles are definitely poisoned and most of them die in the lymph channels and small lymph nodes of the lung. Other dusts including silicates, with the exception of asbestos, have not this action. It is only silica, that can, as far as we know, produce the typical whorl shaped fibrosis which gives the specific nodulation seen in the x-ray films.

Diagnosis:

The diagnosis of silicosis is based on, *first,* occupational history. There must be a history of exposure to hazardous silica dust for a substantial period of time. We have seen a few cases in the British Columbia mines where the exposure was less than three years, all of them acute cases, but the large majority of our cases have been exposed for five years or more. The shortness of exposure in some cases in explained by large concentrations of pure silica dust, but this is not the whole story. There seems to be, in some workmen, a greater susceptibility to silica fibrosis than in others. It has been found that even in rabbits some strains will become silicotic much more readily than others.

The *second* standard of diagnosis is the typical nodular fibrosis seen in the x-ray film. Without this typical nodulation one cannot make a diagnosis of silicosis. It is true that the lungs may be "effectively occupied" by silica dust and the man be removed from contact with the dust for a period of years before the typical nodulation shows.

Two soldiers who were miners prior to the war, both in the same mine, enlisted in 1941. Their enlistment films showed normal chests; yet in 1944 they both showed very marked silicosis, although during the period of three years they were in the Canadian Army Overseas they were not exposed to any dust. The typical case may go on for a number of years and the annual x-ray examination show no change. Gradually, however, there develops a generalized increase of the linear lung markings and some enlargement of the hilar shadows. The linear markings gradually extend to the periphery, showing a fine reticulation. There may be no change in this appearance for a number of years. Irvine of South Africa has likened the appearance at this time to the branches of a leaf-

less tree—then rather suddenly, often in the space of six months or a year, the tree suddenly starts to leaf out—the leaves in this case being the discrete nodules, from 2 mm. to 6 mm. in diameter, widely spread over both lungs. At this time, there are usually few or no symptoms, except possibly, slight dyspnoea on heavy exertion. The disease may remain stationary at this stage for a number of years and the man can carry on at his work quite efficiently. Other cases may progress fairly rapidly, the discrete shadows becoming conglomerate, and showing evidence of silicosis with infection. The man becomes dyspnoeic, develops chronic cough with occasional blood spitting, and is only able to carry on at light work. If he develops manifest tuberculosis with a positive sputum, he rapidly becomes totally disabled and his life expectancy is reduced to a few years.

As to whether a given case will progress rapidly cannot be predicted; and as to why some do and others do not is one of our unsolved problems. Infection with tuberculosis certainly speeds up the progress of the disease, but a number of definitely acute cases of silicosis proceed rapidly to death without any evidence of tuberculosis.

In most cases the progress is slow. There is progressive increase of the fibrosis with compensatory emphysema; dyspnoea becomes quite severe on very little exertion. The man develops an irritative cough, some times with considerable wheeziness. His general health remains good but he cannot earn a living because of shortness of breath. Without serious intercurrent infections these men may live out their life expectancy, especially if they are economically provided for by some form of compensation.

Differential Diagnosis:

It must be laid down as a dictum until further research proves the contrary that, unless a patient has the typical x-ray appearance of nodulation, silicosis cannot be diagnosed as a cause of disability. All the symptoms of silicosis may be present, with also sufficient history of hazardous exposure to dust. Emphysema, chronic heart disease with congestive failure, and asthmatic bronchitis all may produce similar clinical symptoms, and at times the x-ray appearance may be misleading. These cases are the ones that are often most difficult to decide in regard to compensation. There are a number of conditions which show a nodular x-ray appearance that are not due to silicosis.

Treatment:

The reduction of the incidence of silicosis in hazardous industries is a matter of dust control. Proper ventilation, the use of

water, and above all adequate inspection of the mines by competent dust engineers, who not only can find the hazardous areas but have power to see that these hazards are corrected, are the first requisites to the control of silicosis. Aluminum dust used as a prophylaxis is likely to become useful and may be the additional factor that will make our future miners free from silicosis.

As far as the silicotic is concerned, there is nothing as yet that has proven of value. Aluminum dust has been shown to be effective in preventing silicosis in rabbits and also in arresting silicotic fibrosis when given in high doses. A short summary of the use of aluminum dust in prophylaxis and treatment may be of interest.

Use of Aluminum Dust in Prophylaxis and Treatment of Silicosis

In 1937, Denny, Robson and Irvine reported that in dusting experiments with quartz dust they could produce nodular fibrosis in rabbits, but if the quartz dust was mixed with a freshly ground aluminum powder in particles the size of less than 5 microns, with a proportion of aluminum dust of 1 per cent it would prevent the occurrence of nodular fibrosis.

In 1939, they showed this inhibiting action of aluminum is produced largely by covering the quartz particles with an insoluble and impermeable coating which is gelatinous hydrated aluminum, and which stains red with aurine. Further experiments, Belt and King showed the particles so treated were treated by the body cells as more or less inert bodies. Further work by several observers have shown that aluminum dust did not cause lung damage nor did it favour the development of tuberculosis or any other lung condition.

A few papers have come out of Germany describing harmful effects of aluminum powder. There have been some reports that hydrated aluminum has some effect in aggravating an existing tuberculosis. So far there has been no work done that is in any way convincing of this effect.

Following the publication of these beneficial effects of aluminum dust in rabbits, under the supervision of the McIntyre Research Ltd., prophylactic treatment has been installed in a large number of mines in Canada and other places. During the past two years most of the mines in British Columbia have installed this treatment. In the change room, or dry room, there has been installed a blower which blows from small canisters of aluminum powder, giving a concentration of 1 gram of aluminum dust per 1,000 cubic feet of room volume. This is inhaled by the men for a period of 10 minutes before going underground. The treatment is voluntary on the part of the men but has been enthusiastically adopted and

no bad effects have been noted. In fact, in some of the mines it has become quite popular as a cure for various respiratory complaints, and I have been told that some of the women are taking it for a variety of conditions. As to its effect in prevention of silicosis, there is no evidence as yet, nor is it likely that definite evidence can be provided for five to ten years. In no sense does the use of aluminum supplant the other important measures of dust control. In fact, more emphasis has been placed upon these measures.

In 1944, it was decided by the Workmen's Compensation Board of British Columbia to offer to certain pensioners from silicosis the opportunity of taking aluminum dust as a therapeutic measure for their disease. Under supervision of the McIntyre Research an air-tight chamber, 560 cubic feet capacity, was constructed. Into this chamber was blown aluminum dust from small canisters, to give a concentration of 0.5 mgms. of aluminum powder per litre of air. The patients stood outside the chamber and inhaled through the mouth this aluminum dust for a period of ten minutes daily for one hundred treatments. The men selected were all definite cases of silicosis, who had developed no evidence of pulmonary tuberculosis. They were all pensioned from 50 to 100 per cent for disability due to silicosis. They were all assured that any improvement in their condition would not result in reduction of pension. They were all medically examined and x-rayed, both before and after treatment.

In giving results, the matter of improvement or no improvement is based on statement of the workman. In only two have there been changes for the better in the x-ray films. In the rest, there has

RESULTS OF ALUMINUM DUST THERAPY
IN SILICOTIC PENSIONERS

Total starting treatment	36	
Total completing 100 treatments		15
Total completing less than 100 but 50 or more		8
Total completing less than 50 treatments		9
Total still under treatment or not reported		4
Results in 23 who took 50 treatments or more:		
Improved		13
Unimproved		6
Worse		4

Of these 23 — 10 were on 100 per cent pension.
10 were on 60 per cent pension.
3 were on 50 per cent pension.

been no change. There has not been any evidence of progression. While some of the patients stated they were worse, we have noted no harmful results. It has been found that at the beginning some of the workmen had increase of cough for a few days but this did not persist. The psychological effect has been good and the morale greatly improved.

It is impossible to come to any conclusions from such limited numbers. The statement of improvement was entirely symptomatic, and similar results might be obtained by giving any harmless treatment with a large dose of hope and faith. However, generally speaking, the psychologic effect was good on at least fifty per cent. We did not think there was any harm done even in those who stated they were unimproved or worse.

As regards the future use of aluminum dust, we are of the opinion that it does no harm. As a prophylactic its position has been well established by animal experiment. It will be some years before we can assess its full value in humans. As a therapeutic measure its use at present is entirely experimental, and a great deal of animal experimentation remains to be done before it can be accepted generally as of any more than psychological value in the treatment of silicosis. Aluminum cannot in any way supplant as a prophylactic the most important measures of dust control.

The Silicosis Problem In B. C.

In normal times there are from 4,000 to 5,000 men employed underground in the hard rock mines of British Columbia. Up to 1936 it was not a compensatable disease and as far as prevention of silicosis was concerned there was no adequate inspection of the dust and ventilating conditions in the mines. There had been a law preventing dry drilling and also some regulations as kept down dust by water, but generally speaking conditions were very hazardous.

In 1935, a survey was made of five of the leading metalliferous mines (Table I)—to obtain a cross section of the prevalence of silicosis.

All the silicotics were miners who had worked five years or more

TABLE I

Total Number Examined	1,339	
		Per cent
Negative Findings	1,224	91.41
Simple Pulmonary Tuberculosis (All but one of these had worked less than five years underground)	10	0.75
Silicosis	105	7.84
	1,339	100.00

underground. Total number of miners examined who had worked five years or more underground, 703. Of that number 105, or 14.9 per cent, were silicotics.

Tuberculosis was a probable complication in 3 of the stage 3 cases of silicosis (Table II).

TABLE II

Summary of 1,339 Men Examined

Years underground in Metal Mines	Negative	Simple Pul. Tbc.	Stage of Silicosis 1	2	3
1 yr. or less	273	4			
1 to 5 yrs.	354	5			
5 to 10 yrs.	327	1	16	3	
10 to 15 yrs.	207		27	9	2
15 to 20 yrs.	31		7	5	3
20 to 25 yrs.	20		8	11	
25 to 30 yrs.	10		7	2	2
30 to 35 yrs.	2		1	1	1
	1,224	10	66	31	8

SUMMARY

Out of 703 miners who had been five years or more underground, 105 or 14.9 per cent were silicotics; 62.8 per cent of these were Stage 1, 37.2 per cent were showing disability.

Following this survey, silicosis was made a compensatable disease and it was made compulsory for mine owners to have their employees going underground given a yearly examination with x-ray film of the chest. At the present time any workman who has been exposed to silica dust in the mines of British Columbia for a period of three years, or for a lesser period if not exposed outside of British Columbia, is eligible for compensation for disablement due to silicosis.

Silicosis as defined under the Act "shall mean a fibrotic condition of the lungs caused by dust containing Silica and evidenced by specific x-ray appearance, accompanied by a substantially lessened capacity for work."

The regulations regarding annual examination of employees are that each employee must have a medical examination and x-ray film of his chest, and a certificate for fitness for underground work issued within two months of the time he starts working underground, and this examination must be repeated annually.

These examinations are made by the mine doctors at the expense

of the employer. The examination forms and x-ray films are forwarded to the Workmen's Compensation Board and reviewed there by the Silicosis Referee who may cancel or re-issue any certificate if he considers it advisable.

The policy in regard to issuing certificates is that all cases of suspected active tuberculosis shall be excluded.

Cases of silicosis showing progression or complicated by tuberculosis are advised to apply for compensation and removed from underground work.

Cases of early silicosis which are not showing progression, especially in the older men, are issued certificates if they wish to continue. We have not found that these cases progress substantially faster when removed from underground work than if allowed to remain at work to which they are accustomed. Compensation is based on disability and it is an economic disaster to remove men from work they have been doing for fifteen or twenty years and ask them to make a living at something else. We consider also that most of the mines of British Columbia are now reasonably safe.

Besides compensation and yearly examinations, in September 1937, the Workmen's Compensation Board appointed a competent dust inspector to visit the mines at regular intervals, and to make dust counts and recommendations regarding improvement of ventilation. A second man has recently been appointed. Under their recommendation many hundred thousands of dollars have been spent on improving the dust conditions. They report that in most mines conditions are reasonably good. Their standard is 300 particles of dust or less per cc. of air. In some parts of the mine, as in the raises where the men are drilling, conditions are still hazardous, counts being sometimes as high as 2,000 particles per cc., so that it is not possible as yet to do away entirely with hazardous dust.

It is our impression, not as yet confirmed by accurate statistics, that there is a definite fall in recent years in the incidence of new cases of silicosis. It must be remembered that at the time this disease was made compensable, there were a large number of miners who had advanced silicosis, either working or who had just stopped working. There were also a large number of men who had already been exposed for four or five years to old conditions prevailing before 1937. It is therefore going to be some years yet before we can estimate the benefit being derived from general dust prevention measures, as well as the use of aluminum.

RESUMEN

De 703 mineros que habían trabajado subterráneamente cinco años o más, 105, o sea el 14.9 por ciento, eran silicóticos; el 62.8

por ciento de estos se encontraban en el Período 1, mientras que el 37.2 por ciento sufrían incapacidad.

Subsiguiente a este censo se declaró que la silicosis sería una enfermedad indemnizable y se obligó a los dueños de las minas a que sometieran a exámenes anuales con películas radiográficas del tórax a todos sus empleados que trabajaban subterráneamente. Al presente, todo trabajador que ha estado expuesto al polvo silícico en las minas de la Columbia Británica por un período de tres años, o por un período más corto si no se ha expuesto fuera de la Columbia Británica, es elegible a obtener indemnización por incapacidad debida a silicosis.

De acuerdo con la definición de la Ley, por silicosis "se da a entender una condición fibrosa del pulmón causada por polvo que contiene Sílice, y evidenciada por el aspecto radiográfico específico, acompañada de una disminución substancial de la capacidad para el trabajo."

Los reglamentos relativos al examen anual de los empleados exigen que todo empleado debe ser sometido a un examen médico y a una película radiográfica del tórax, y que debe obtener un certificado de aptitud para el trabajo subterráneo expedido dentro de dos meses, a partir de cuando comienze a trabajar subterráneamente, y que se debe repetir anualmente este examen.

Los doctores de la mina hacen estos exámenes a costa del patrono. Los resultados de los exámenes y las películas radiográficas son remitidas a la Junta de Indemnización de Trabajadores donde son revisadas por el Arbitro de Silicosis, quien puede cancelar o re-expedir cualquier certificado si lo considera prudente.

El plan de acción en cuanto a la expedición de certificados, es que se debe excluir a todos los casos sospechosos de tuberculosis activa.

A los casos de silicosis que manifiestan progresión o que están complicados por tuberculosis se les recomienda que soliciten indemnización y se les retira del trabajo subterráneo.

A los casos de silicosis precoz, que no manifiestan progresión, especialmente en los hombres más viejos, se les expiden certificados si desean continuar trabajando. Hemos notado que estos casos no progresan con menor rapidez cuando se les saca del trabajo subterráneo que cuando se les permite que continúen en ese trabajo a que están acostumbrados. La indemnización está basada en la incapacidad y es un desastre económico el sacar a estos hombres del trabajo que han estado haciendo por quince o veinte años y obligarlos a ganarse la vida en otra forma. Consideramos, además, que dele la mayor parte de las minas de la Columbia Británica están ahora a salvo.

Además de las indemnizaciones y de los exámenes anuales, la Junta de Indemnización de Trabajadores nombró a un competente inspector de polvo, en Septiembre de 1937, para que visitara las minas a intervalos regulares, y para que hiciera estudios del polvo y ofreciera recomendaciones con el objeto de mejorar la ventilación. Recientemente se ha nombrado a otra persona más. Siguiendo sus recomendaciones se han gastado muchos cientos de miles de dólares para mejorar las condiciones de polvo. Informan ellos que en la mayor parte de las minas las condiciones son razonablemente buenas. Su norma es de 300 partículas de polvo, o menos, por cc. de aire. En algunos lugares de la mina, como en los levantamientos donde los hombres están taladrando, las condiciones todavía son peligrosas, pues las cuentas a veces ascienden a 2000 partículas por cc., así es que todavía no es posible eliminar por completo el peligro del polvo.

Nuestro parecer, no confirmado todavía por datos estadísticos exactos, es que en los años recientes ha tenido lugar una disminución bien definida en la frecuencia de nuevos casos de silicosis. Debe recordarse que cuando se declaró que esta enfermedad sería indemnizable, existía un gran número de mineros que padecían silicosis avanzada que todavía estaban trabajando o que acababan de dejar de trabajar. Existía también un gran número de hombres que ya habían estado expuestos por cuatro o cinco años a las anticuadas condiciones que prevalecían antes de 1937. De manera pues que no será sino dentro de varios años cuando podremos calcular el beneficio derivado de las medidas generales para eliminar el polvo, así como del empleo del aluminio.

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Pneumococcus Pneumonia and Its Complications*

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The subject of this essay is pneumococcus pneumonia and its complications as seen in San Francisco over various periods of study from December 1932 to June 1946.

Pneumonia has been ever present in San Francisco though it has been of varying severity and etiology from year to year. Until the 1918 epidemic of influenza and the pneumonias which complicated it, pneumococcus pneumonia is said to have been much more common than in the years which followed the first world war. It is also said that the respiratory flora in the years following the first war was such that most of our serious respiratory infections were due to organisms other than the pneumococcus and many of them were largely concerned with the streptococcus. It was also believed that lobar pneumonia of pneumococcus origin was considerably less prevalent in California than in the Eastern United States and in the midwestern states. Since 1931, there has been a return to the usual varieties of pneumonia and it is apparent now the severity of the disease is essentially the same here as in other parts of the United States though the disease is still probably not as frequently encountered as on the eastern seaboard. Marked variations of morbidity and mortality may occur from year to year, as shown by the paucity of pneumonia in San Francisco during the most recent eleven month's period.

The purpose of this paper is to discuss pneumonia in this area over three widely separated periods starting in 1932. The first study was made at the San Francisco Hospital from December 1932 to June 1933. The tabulation of figures of this period with the figures of the complications, results of treatment, etc., will be followed by the description of a second period in which specific therapeutic agents were used, and which show improvement in the mortality rate; and the last period which shows results of more efficient therapeutic combinations.

In the early figures for 1932 and 1933 the general consideration is about as follows: The average age of this first group was 42 years. The eldest 77, the youngest 5. Males and females were affected in the ratio of 3 to 1. When one remembers this is a city hospital group, one must know that complicating disease was high

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and the commonest chronic ailment was circulatory disease as manifested by hypertension and congestive failure, coronary artery disease, and abnormal cardiac rhythms. Complicating factors were such things as syphilis, tuberculosis, malignancy, alcoholism, etc. With all of these factors taken into consideration, the mortality for the 1932-33 period was approximately 42 per cent. With those individuals who were unfortunate enough to have a positive blood culture on entry, the total mortality was 60 per cent; while of those who had negative blood cultures 19 per cent died.

For the period December 1932 to June 1933, during which time 250 individuals with pneumonia were seen, empyema occurred 7 times with 4 dead and 3 recovered. Pericarditis occurred 5 times, 3 died and 2 recovered. Two of these who died with pericarditis, also had empyema. Pleural effusion without empyema occurred on 31 occasions. On 2 of these occasions, streptococcus was found in the pleural fluid and in the remainder the pleural fluid was sterile. Of these individuals, 9 or approximately 29 per cent died and 22 or 71 per cent survived. Known endocarditis occurred in only 1 of this group of 250 and that individual died; while meningitis occurred 4 times with 4 dead, in each instance due to pneumococcus type IV. Lung abscess occurred 4 times, 2 of these individuals died and 2 recovered. One of these with lung abscess who died also had empyema. Thus it will be seen that there were 52 complications in 49 patients,—that is approximately 20 per cent of the patients in this early series showed complications of a serious nature.

The treatment of this first group was confined to nursing, oxygen, increased fluid intake, the use of alcohol in those chronically addicted to its use, and in a small number specific antiserum was used; and in those individuals who had serious abdominal distention, an increased amount of sodium chloride was used to combat this complication.

The second group of cases comprises 250 patients with pneumococcus pneumonia seen in the same hospital in the 2 years from July 1943 to July 1945. The age range and complicating disease was approximately the same as in the earlier group. It is interesting that in this group those with negative blood cultures had a mortality rate of approximately 11 per cent, while those with positive blood cultures had a mortality rate of 42 per cent. Total mortality rate for the two years was approximately 16 per cent, the total rate being 26 per cent in 1943-44 and 13 per cent in 1944-45.

The treatment of this group in addition to the routine noted above, consisted in sulfadiazine which was used in 70 per cent of the individuals treated and sulfamerazine in 18 per cent, and a few patients only were treated with sulfathiazole. All of these sulfa

drugs were used in the routine fashion with an initial dose of 4 grams followed by 1 gram every four hours of sulfadiazine and 1 gram every 8 hours in the case of sulfamerazine. Those patients who appeared to be in serious condition were given the sodium salt of the sulfa drug in initial dose of 5 grams in 1,000 cc. of lactated Ringer's solution. In addition to this, due to high mortality rate of type VII those individuals with positive blood cultures of type VII were likewise given rabbit serum in doses of about 200-300,000 units.

This group showed 41 complications as previously listed or a really higher rate of complications than in the group of 1932-33; empyema heading the list with 13 cases with 3 deaths or an empyema mortality of 23 per cent compared to the higher mortality of 57 per cent in 1932-33. Let it be pointed out here that the number of cases involved is far too small from which to draw accurate conclusions. All those individuals with endocarditis and lung abscess died. These, of course, remain the most serious complications of pneumococcus infection. In the second period, 1943-45, 16 deaths were attributable to what we choose to call "peripheral vascular collapse." This cause of death was not listed in our earlier figures, due to failure of classifying deaths in this category. In the 1943-45 period 18 individuals were seen who, we felt, were in this category, and all but 2 died, or 89 per cent.

When this study was undertaken, it was thought that for the period July 1945 to June 1946 we would have commensurate figures to present, but the year failed to produce a great number of pneumonias so that for this last period, 1945-46 we have only 45 pneumococcus pneumonias to report. In addition to there being but few cases on our service this year, the pneumococcus pneumonias were also quite mild, there being only 4 deaths among the 45 patients or a mortality of about 9.5 per cent. As further evidence of the mildness of the infections, only 18 had positive blood cultures or a positive blood culture rate of 40 per cent, but 3 of the 4 deaths recorded were among those with positive blood cultures, the positive blood culture mortality thus being 16 per cent, or the lowest positive blood culture mortality rate which we have recorded. Let it be again noted that the numbers are too small for accurate statistical data. Of those individuals who failed to show a positive blood culture only one died, and that one was moribund on arrival on the ward.

The complications, including so-called peripheral vascular collapse numbered only 12 or about 28 per cent, but queerly enough among the patients with complications, only one person died, and he was the single one who had peripheral vascular collapse. None of the 5 persons with empyema, the 5 with pleural effusion, or the

1 person with lung abscess died. In this last eleven months there was no case of either meningitis or endocarditis. One individual who had a positive blood culture with type VIII pneumococcus, and who also had an empyema was treated with sulfadiazine, penicillin, and rabbit antiserum, and survived.

It seems almost without purpose to break down further the figures of the last eleven months, since the cases are too few for accurate statistics, yet it may be mentioned that of the 18 persons treated with sulfadiazine or sulfamerazine alone, none died. Obviously they responded well to this drug, or in these days they would have had further help from both penicillin and/or serum. We believe it should also be mentioned that those persons seriously ill *were* treated with sulfonamides and/or penicillin. Twelve persons were treated with penicillin alone and two died, both of whom were moribund on entry to the ward; one being unconscious on entry, living only 7½ hours and the other, aged 63 with a leucocyte count of 4,600 per c.m.m. lived less than 48 hours. Fifteen persons seriously ill were treated with both penicillin and sulfadiazine, 2 of whom died, both having positive blood cultures and one having had several ribs broken before entry, and both of whom were seriously alcoholic and developing delirium tremens in the hospital. The person with the broken ribs survived eleven days. Any recital of statistics on pneumonia is useless unless some conclusions may be drawn, as to the cause of the variation in the figures presented.

All of the patients presented in these three groups, as well as all of those seen in the interim years not reported had certain basic therapeutic procedures used upon them. These consisted of bed rest, nursing care, fluids orally or parenterally, liquid of soft diet, alcohol when indicated, sedatives and drugs for relief of pleural pain, surgical relief for collections of fluid in the chest, salt restoration and whatever drugs were indicated for the cardiac conditions arising during the disease or present on entry.

In the early years the use of specific serum was a factor in the treatment, but not a great one with us even though the mortality rate was somewhat lowered by its use.

In the years after 1938 sulfonamides were used almost universally, with definite reduction in the mortality rate, but not until 1942 were they used in all cases, so that the 1943-45 period was a good one in which to estimate the mortality rate with this therapy; and of course, this was with a great reduction from the earlier years. It is our impression, that the use of these drugs alone is responsible for the saving of many lives in this disease and even though the *rate of complication is not lowered by the use of these drugs* the mortality from complications is lowered.

It is also our impression that if sufficient dosage of these drugs can be maintained over a sufficient length of time, certain of the complications such as pneumococcus meningitis which were almost hopeless before the advent of the sulfonamides may be favorably influenced, while the combination of sulfa drugs and penicillin together, wisely used, may also *prevent* some of the serious complications or at least make them less serious.

Our present ability to go directly to the seat of trouble in empyema or meningitis with such a potent drug as penicillin was the dream of our forbears, and only since the advent of penicillin is this possible. Three of the empyemas seen in 1945-46 (total seen, 5 in number) were treated by the closed method, i.e., by tapping one or more times and instilling penicillin directly into the chest cavity in doses of from 50,000 to 100,000 units, and in each instance cessation of the empyematous condition resulted. Recurrent drainage by needle or trocar was all that was necessary in 2 other cases.

Pleural effusion, which may or may not have resulted in empyema under earlier conditions, so called rather than empyema because of failure of growth of organisms from the chest fluid, was present 5 times and treated with instillation of penicillin 3 times, with cure on two occasions without further treatment, and with further drainage in one instance. On two other occasions, small amounts of pleural fluid were evacuated without the necessity of further tapping, as the fluid was sterile on culture and did not recur.

SUMMARY

1) Statistics on a relatively small number of cases of pneumococcus pneumonia in San Francisco have been presented. While our statistical data are small, it is in accord with the figures presented in larger series.

2) There has been a constant reduction in mortality rate consistent with the introduction of newer therapeutic agents.

3) Complications may be less and certainly the consequences of complications are less severe with the employment of the newer drugs.

4) The high mortality rate formerly accompanying the positive blood culture have been drastically reduced by the use of these newer drugs.

5) We must not overlook the fact that marked variations in the severity of infection and variation in morbidity influence the statistics in pneumococcus pneumonia to a large extent.

We acknowledge with thanks the aid of the resident staff and visiting staff of the University of California Medical Service at the San Francisco

Hospital, particularly Doctors George Warner and Frank Gardner for their aid in the preparation of the statistics presented.

We likewise acknowledge the cooperation of the Director of Public Health of San Francisco, without which this study would not have been possible.

RESUMEN

1) Se han presentado datos estadísticos sobre un número relativamente pequeño de casos de neumonía neumocóccica en San Francisco. Nuestros datos estadísticos, aunque son pocos, están de acuerdo con las cifras presentadas en series más amplias.

2) Ha tenido lugar una reducción constante en la mortalidad, consistente con la introducción de agentes terapéuticos más modernos.

3) Puede haber menos complicaciones e, indudablemente, las consecuencias de estas complicaciones son menos graves cuando se emplean las drogas más modernas.

4) La alta mortalidad que antes acompañaba a los casos en los que el cultivo de la sangre era positivo ha sido reducida en extremo con el uso de estas nuevas drogas.

5) No debemos pasar por alto el hecho de que marcadas variaciones en la gravedad de la infección y variación en la morbicidad influyen en sumo grado los datos estadísticos referentes a la neumonía neumocóccica.

Lung Abscess (Surgical Aspects)*

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I wish to make this presentation as succinct and practical as possible. It will, therefore, be necessary to pass over many aspects of the general hygienic care, adequate diet, vitamin administration, bacteriological findings and etiology, which are of importance in a general discussion of pulmonary abscess. Suffice it to say, it is the belief of most of us that pulmonary abscess, in general, is a disease of aspiration rather than an embolic disease.

I have long been opposed to the tendency of many physicians to separate the management of lung abscess into medical treatment for one period of time and surgical treatment for another. In the not too far distant past it was the custom to set a time limit, usually from six weeks to three months, for the period of purely "medical treatment." If the abscess was not cured by then, the case automatically became surgical. The effects of treating such a serious disease by any similar routine have been amply reflected in the appalling mortality statistics of past years. While it has been estimated that from 25 to 35 per cent of patients with pulmonary abscess become well spontaneously, the mortality has been from 30 to 50 per cent in most series of prior years. The communications of Neuhof, Overholt and Rummell, Betts and others have demonstrated that such a mortality can be reduced. If the abscess is to be treated definitively, it should be done during the acute phase. Within six weeks from the onset chronic changes, pathologically, already have become established in the pulmonary parenchyma and the optimum time for a cure is past.

There is now, I believe, greater appreciation of the fact that pulmonary abscess is a very unpredictable disease with ever-present surgical potentialities, and that the best results are obtained by the physician and thoracic surgeon working as a close team from the onset.

Pulmonary abscess has the characteristics of abscess elsewhere in the body in that cure is dependent upon adequate drainage. Because of the bronchial connection in all pulmonary abscesses it is possible that so-called internal drainage may suffice. I would,

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therefore, rather speak in broad terms of the drainage aspects of pulmonary abscess rather than of the purely surgical aspects since an appreciable percentage of abscesses may be cured by improving internal drainage.

As a general working rule and without specifying too closely concerning bacteriology, the odor of the excretions from a pulmonary abscess gives some index as to how early or how necessary external drainage, i.e., surgery, may be. A more putrid odor usually means a higher percentage of anaerobic organisms. In such abscesses it is usually more certain that early external drainage will be necessary.

The use of chemotherapy or antibiotic therapy must not be minimized in the treatment of abscesses. It has been suggested, moreover, that the combination of a sulfa preparation and penicillin may be more efficacious than either drug used alone. Such treatment can be overdone, however, and it must always be remembered that adequate drainage is necessary regardless of how many grams of sulfadiazine or how many thousand units of penicillin are poured into the patient. It is possible that the abscess cavity even may be temporarily sterilized although a slough remains. This still necessitates external drainage, regardless of the fact that organisms are absent for the time being.

Once the patient with pulmonary abscess has come under the physician's care, improvement must be prompt and progressive or the course of treatment should be radically changed. This improvement must be both clinical and roentgenological. No abscess is doing well which remains unchanged on the roentgenogram for two or three weeks at a time even if there has been an amelioration of symptoms.

As has been stated above, the essential treatment of pulmonary abscess is to improve drainage. Internal drainage is promoted by postural drainage and by bronchoscopic aspiration. Postural drainage, unless it is done carefully, may produce more harm than good. Many of these patients are acutely ill and cannot stand the physical exertion of routine postural drainage. The type of postural drainage which should be used must be individualized. In general, the patient should be instructed to assume the position which makes him cough most, four or five times during the day. Cough is a natural defense mechanism on the part of the body in raising secretions and is a good index as to whether or not postural drainage is being done in the most efficient position. The usual position of postural drainage with the patient hanging over the bad almost straight down from the hips may be harmful if the abscess is in the middle or upper lobes.

It is obvious to those of us with experience that the bronchoscope

is an invaluable means of improving internal drainage in many cases of pulmonary abscess. In the past bronchoscopy has been used far too infrequently during the acute stage. Bronchoscopy should be one of the earliest therapeutic procedures employed. It has been stated that many of these patients are too sick to be subjected to bronchoscopy. Rather it should be said that they are too ill not to be bronchoscoped. There are many who are still unconvinced as to the value of bronchoscopy in pulmonary abscess. I am certain that such physicians either have not had the benefit of an efficiently performed bronchoscopy, or, they have been swayed by the bronchoscopist's enthusiasm and allowed repeated bronchoscopies (as many as fifteen to twenty) to be performed without appreciable improvement in the patient's condition. This cannot be construed as an indictment against the method but only an indictment of the bronchoscopist's judgment. The bronchoscope is not an infallible instrument. One must not try to do the impossible with repeated aspirations if progressive clinical and roentgenological improvement does not ensue.

In brief, the bronchoscopist can be depended upon to do the following tasks: (1) Remove obstructing inspissated excretions from the main and secondary bronchi; (2) Make an occasional diagnosis of unsuspected foreign body or neoplasm as the causative agent of the abscess; (3) Elicit selective cough with further evacuation of excretions from the affected lobe; (4) Shrink the congested edematous mucosa which in itself, can cause visible obstruction of the bronchial airway; (5) Pass long curved aspirators well into the smaller bronchi of the diseased pulmonary segment and, rarely, directly into the abscess cavity.

In favorable cases, such as illustrated below, the effects of bronchoscopy can be seen within a few days after the first aspiration. When such improvement occurs it is unquestioned both clinically and roentgenologically. When doubt exists as to whether improvement is taking place, a second or even a third bronchoscopy at five to seven day intervals may be indicated. If there is still a question after seven to fourteen days it is then unlikely that any improvement has occurred and the course of treatment should again be changed. Undoubted clinical improvement is manifested by decrease in the cough, reduction in the amount of sputum, a change in the character and odor of the sputum, a reduction in the fever, and an increased sense of well being on the part of the patient. Should the fever continue at its previous level, or should the secretions remain purulent and foul it is certain the bronchoscopy is not being effective.

In general, brisk hemorrhage contra-indicates bronchoscopy and should be an indication for early surgical drainage. Sudden thor-

acic pain, dyspnea and evidence of rupture of the pulmonary abscess into the pleural cavity are likewise contra-indications for further bronchoscopy.

Rupture of an abscess into the pleural cavity constitutes a surgical emergency. The overwhelming toxicity which develops from the insult of a virgin pleura with necrotic debris and anaerobic infection frequently leads to rapid termination. When faced with such a situation, temporizing with repeated aspirations and the injection of sulfa drugs or antibiotics is almost certain to end disastrously. Likewise closed intercostal drainage of this type of acute empyema is unsatisfactory in the extreme. Such treatment frequently results in a spreading anaerobic cellulitis and fasciitis of the thoracic wall and the mortality is very high. The treatment of choice is to ignore the abscess for the time being and to open the pleural cavity widely by posterior resection of a long length of the eighth or ninth rib. Rapid evacuation of infected contents is accomplished and multiple Penrose drains are introduced into the pleural cavity, supported by a large pack. If, and when, the lung re-expands the abscess can be dealt with as necessary at a later date.

If, within two or three weeks of the onset of the abscess (or at the most six weeks), there has not been evidence of steady improvement external surgical drainage should be undertaken. If performed within this period of time it is probable that changes in the parenchyma due to chronic infection, such as increased scar tissue formation, and bronchial damage with associated bronchiectasis have not yet occurred. In most instances a decision can and should be made long before the expiration of six weeks.

It is generally agreed that most abscesses can be drained by a one-stage technique and this is certainly preferable if it can be accomplished. Since nearly all abscesses present on a pleural surface, adhesions rapidly form between the thoracic wall and the lung. Accurate roentgen localization is necessary prior to operation. Following the removal of sections of one or two ribs it can be easily seen whether or not adhesions exist beneath. Should there be no adhesions some type of irritating pack such as half-strength tincture of iodine or 1-1000 acriflavine solution should be used for a period of approximately seven days between stages. Generally it is wise to recheck with roentgenograms between stages to be sure that one is approaching the abscess properly. The abscess should be opened with a cautery and the contents evacuated simultaneously with suction. The cavity is then packed loosely with gauze and repacked as necessary until healing takes place. In most instances where the abscess is acute and the destruction of lung has not been too great, healing by resolution and by re-

expansion of the remaining lung will take place, with gradual obliteration of the abscess cavity. In some instances the healing process may be delayed beyond six weeks. If the cavity is clean, some type of filling with free fat grafts or with a pedicled muscle flap is advisable.

Treatment of chronic pulmonary abscess presents a considerably

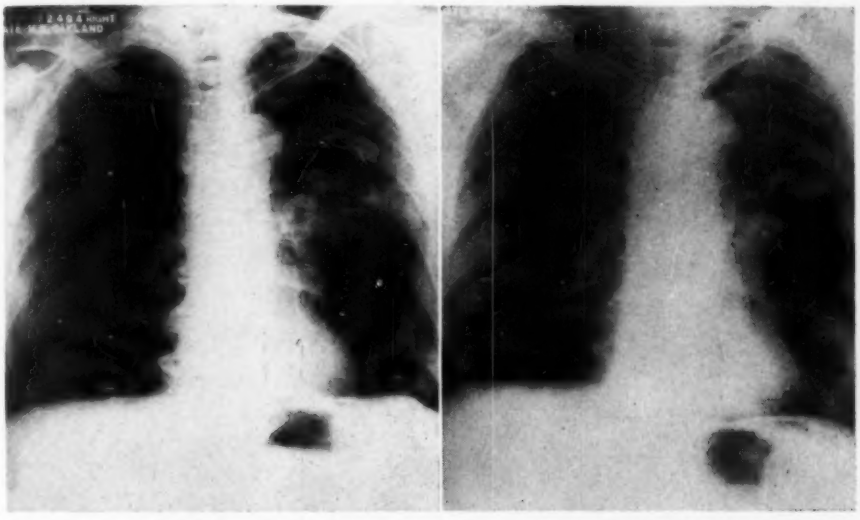

FIGURE 1 FIGURE 2

Fig. 1, Case 1: Frontal roentgenograms showing multilocular abscess in left upper lobe. This film was taken five days before the first bronchoscopy. No essential change in roentgenogram over a period of three months.—*Fig. 2, Case 1:* Roentgenogram taken five days following first bronchoscopy (10 days following Fig. 1). Marked regression of the abscess has occurred. Note the loss of fluid levels and decreased infiltration. The clinical improvement was rapid.

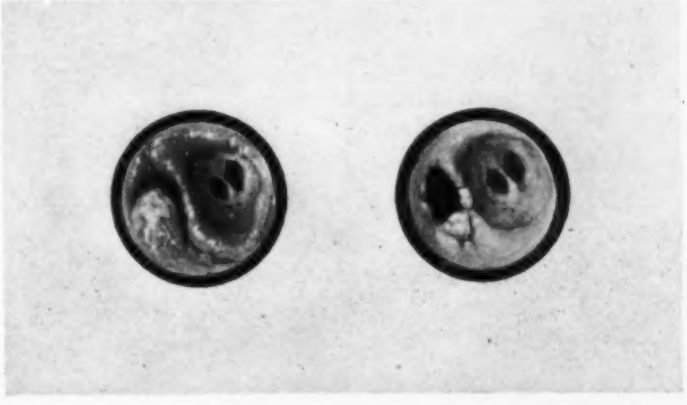

Figure 3, Case 1: (A) Bronchoscopic view of left upper lobar orifice. The partial obstruction is due to tenacious purulent sputum and mucosal edema. (B) Six days later. The mucosal edema and congestion have subsided. A single globule of pus remains. (This figure reprinted from Samson, P. C.: *Am. Rev. Tuberc.*, 38:688, December 1938).

different problem. External drainage of such abscesses is often unsuccessful because of secondary changes in the lung. If they have been drained, the cavity often will not become obliterated, fistulae will remain open and purulent discharge will persist. Prior to operative intervention a complete study of the lung should be done by bronchoscopic examination and bronchograms. If the bronchi show any deviation from the normal or if there is bronchiectasis, lobectomy is by all odds the treatment of choice. Lobectomy performed in the modern manner, carries far less hazard than the external drainage of a chronic abscess.

The following case briefs illustrate some of the points which I have emphasized. The first two cases show the value of bronchoscopy in improving internal drainage. In both cases there is little doubt that bronchoscopy was the deciding factor in returning these patients to health. It will be noted that one case was treated before

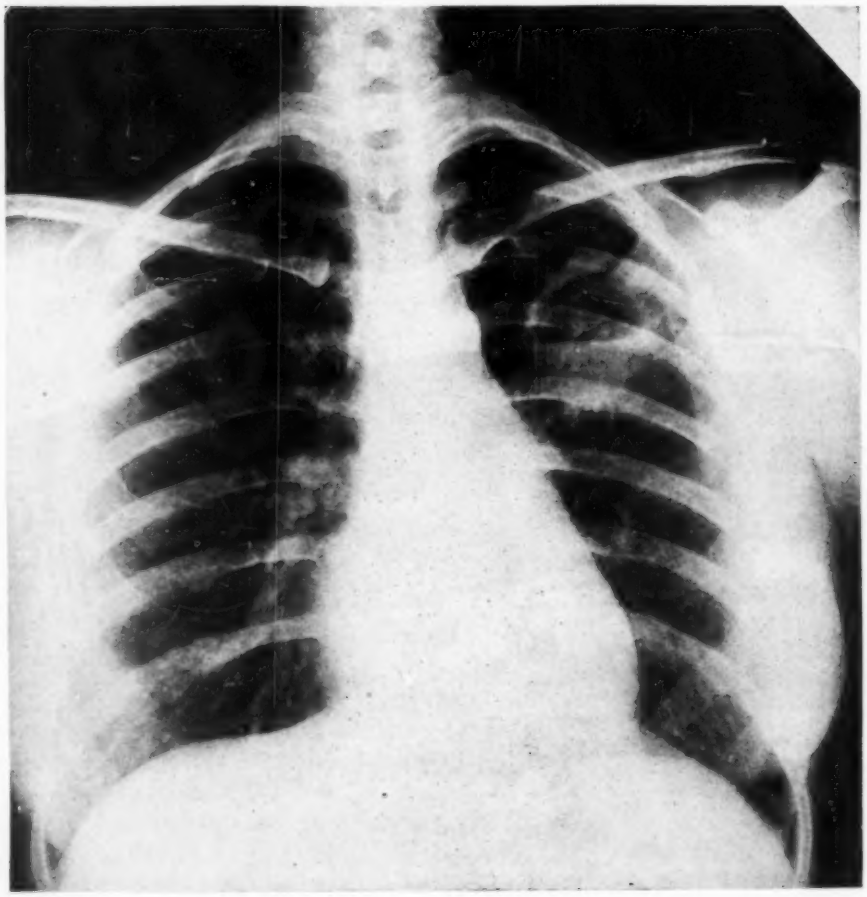

Figure 4, Case 2: Frontal roentgen film taken two days prior to bronchoscopy. There is a 5 cm. cavity in the anterior portion of the left upper lobe which shows a fluid level and considerable surrounding pneumonitis.

the advent of chemotherapy and the second had both sulfadiazine and penicillin in addition to the other treatment.

Case 1: This seventy one year old male had an acute onset three months before I saw him, characterized by a left-sided pleurisy, fever of 102°-103° and a racking cough. Within a short time he was raising 200 to 300 cc. of foul sputum daily. During the three months period he was kept on strict bed rest with a modified postural drainage. There was slight clinical improvement; the fever only peaked at a 101° and he was raising 150 to 200 cc. of foul sputum a day. The roentgenogram, however, had remained essentially unchanged during the three months (Fig. 1). Of course in a man of this age an excavating carcinoma had to be considered. Bronchoscopy was done at once and was repeated six days later. The second roentgenogram was taken five days following the first bronchoscopy (Fig. 2). Four days following the first bronchoscopy the sputum had become odorless and temperature was normal. Four days following the second bronchoscopy all cough and expectoration ceased. Figure 3 shows the bronchoscopic appearance at the time of the two bronchoscopies. It will be noted that in (A) there is partial obstruction of the left upper lobar bronchus from edema and tenacious purulent secretions. This had decreased markedly within six days.

Case 2: This thirty year old female delivered her third child following a difficult and prolonged labor. Gas and oxygen had been given and the patient responded poorly. She was cyanotic and the pulse was irregular. Within two days there was soreness of the left upper anterior chest and the patient developed cough, sputum and fever. The sputum had no

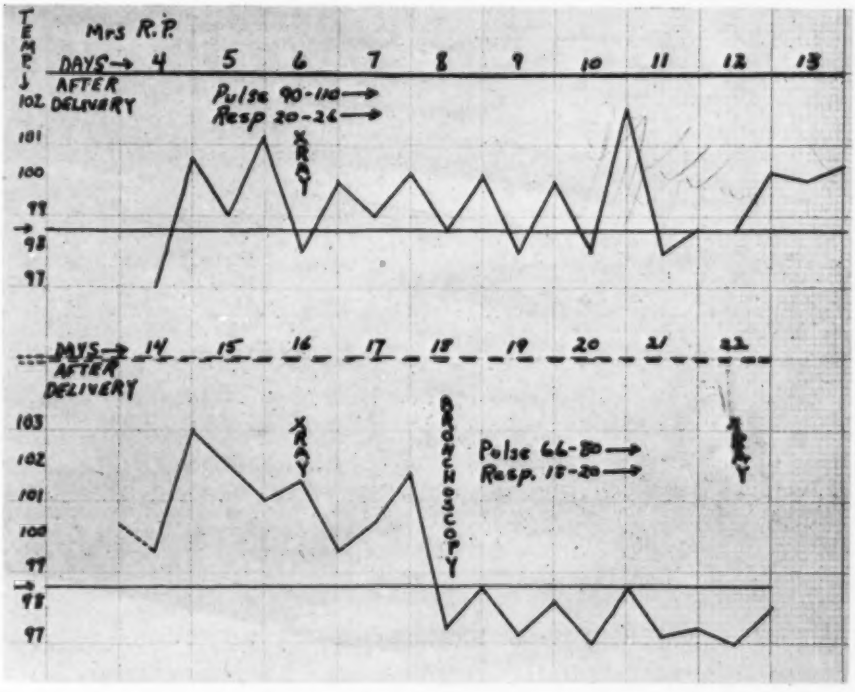

Figure 5, Case 2: Graphic record of temperature. Note immediate subsidence of fever following bronchoscopy.

odor but had a bad taste. She was placed on both sulfadiazine and penicillin therapy. Roentgenograms showed a rounded excavation in the left upper anterior chest (Fig. 4). She was running a swinging fever. Following bronchoscopy there was an immediately subsidence of temperature to normal and within four or five days all sputum ceased (Fig. 5). There was rapid clearing of the abscess in serial roentgenograms (Fig. 6).

As has been emphasized above the patients who do not improve progressively on a regimen consisting of postural drainage, bronchoscopy and chemotherapy should be subjected to surgical drainage within a relatively short period of time, usually in from two to three weeks. This means that surgical drainage will be performed ideally during the acute or subacute stage. It must be constantly kept in mind that merely "holding one's own" is not sufficient.

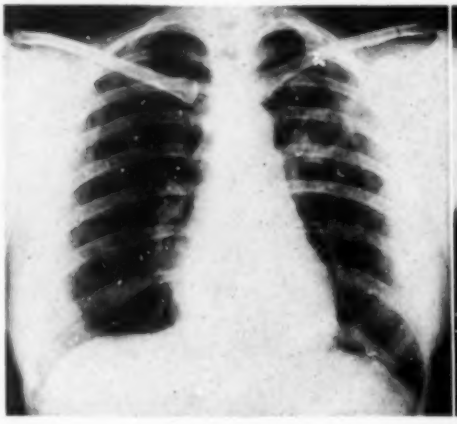

FIGURE 6

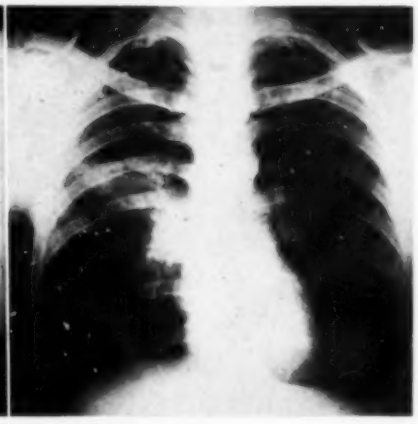

FIGURE 7

Fig. 6, Case 2: Film taken four days following bronchoscopy. Radiologic improvement manifested by a decrease in the size of the abscess, disappearance of fluid level and lessened surrounding reaction.

Fig. 7, Case 3: Roentgen film taken December 11, 1945. Note the area of infiltration in which is a small radiolucent zone. This suggests an abscess cavity partially filled with necrotic debris. Fluoroscopy three days prior to the bronchoscopy on December 26th showed essentially the same picture.

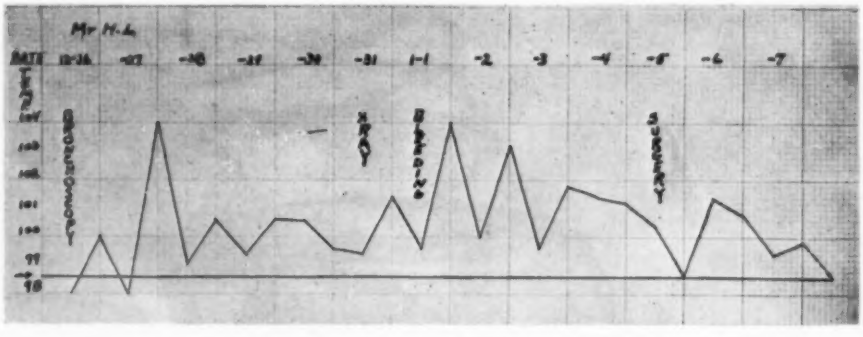

Figure 8, Case 3: Temperature graph following bronchoscopy. It will be seen that the fever continues. The patient's cough and expectoration were unabated.

Insofar as pulmonary abscess is concerned a stationary course is a retrogressive course. Both clinical and roentgen improvement must be obvious. A single case record will suffice to demonstrate the desirability of proceeding with surgery when other methods have failed.

Case 3: This thirty five year old male had a febrile pulmonary illness in August 1945 but apparently recovered from this. Late in November 1945 he developed productive cough with foul sputum and blood spitting. There was loss of weight. He was hospitalized during early December and placed on postural drainages and large doses of penicillin (Fig. 7). There was little improvement and his fever continued. Bronchoscopy on December 26, was followed by increased fever and by an increase in the amount of expectorated blood (Fig. 8). Roentgenograms showed pro-

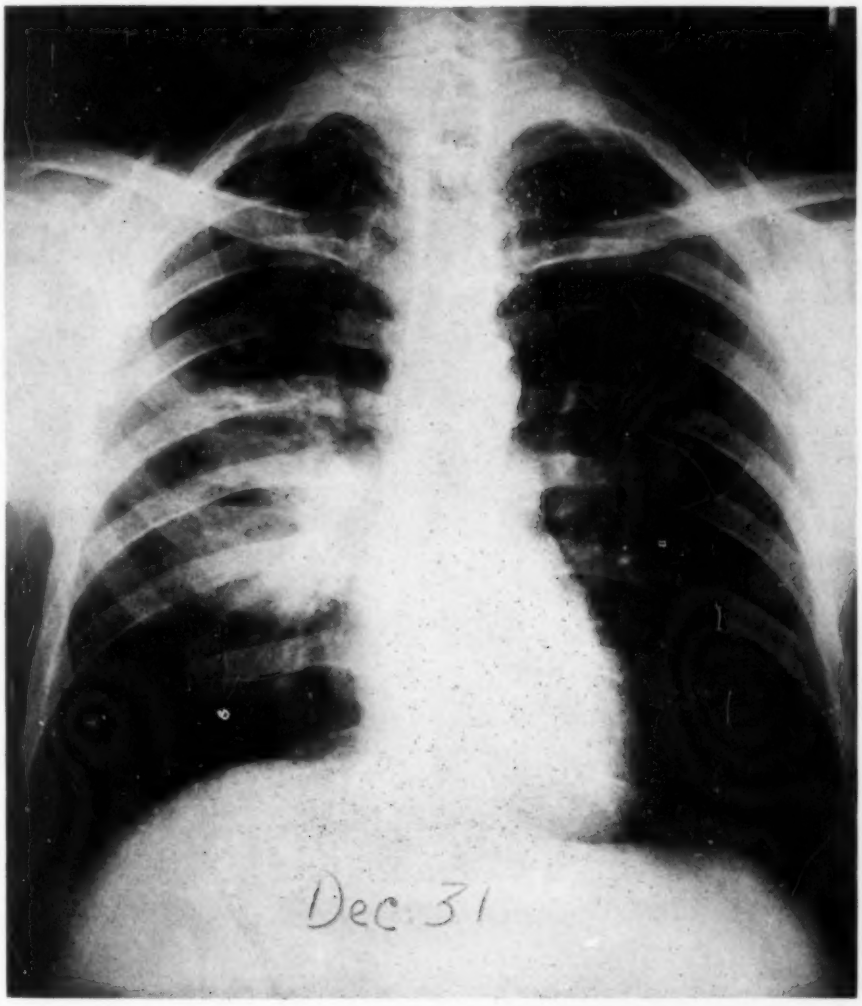

Figure 9, Case 3: This film was made five days following bronchoscopy. There is progression of the infection.

gression of the infection and surgery was instituted within a few days (Fig. 9). After a prolonged convalescence the patient's lung eventually became expanded, the bronchial fistulae sealed over, and the sinus became epithelialized.

In general, the more chronic the abscess the less chance does it have of being cured by external drainage. It is becoming more widely recognized that chronicity brings in its wake secondary changes in the involved lung which may make cure by drainage an impossibility. Occasionally one may be tempted to perform secondary drainage procedures on an abscess which is not "cleaning up" properly. These procedures are frequently unsuccessful because of the underlying irreversible pulmonary damage. In fact, Churchill has stated that no abscess which must be redrained will be cured. Further drainage may be indicated, however, if a secondary acute abscess develops as an extension of the original process.

Many surgeons are convinced that chronic pulmonary abscess should now be treated primarily by pulmonary resection rather than by external drainage. If preoperative survey shows an entire lobe is damaged total lobectomy should be performed. If the disease is *definitely* localized, segmental lobectomy may be indicated. The lingular division of the left upper lobe and the dorsal (or apical) divisions of the lower lobes lend themselves particularly well to segmental lobectomy. If all lobes on one side are involved, total resection by individual ligation is infinitely preferable to attempted cautery pneumonectomy. Even though external drain-

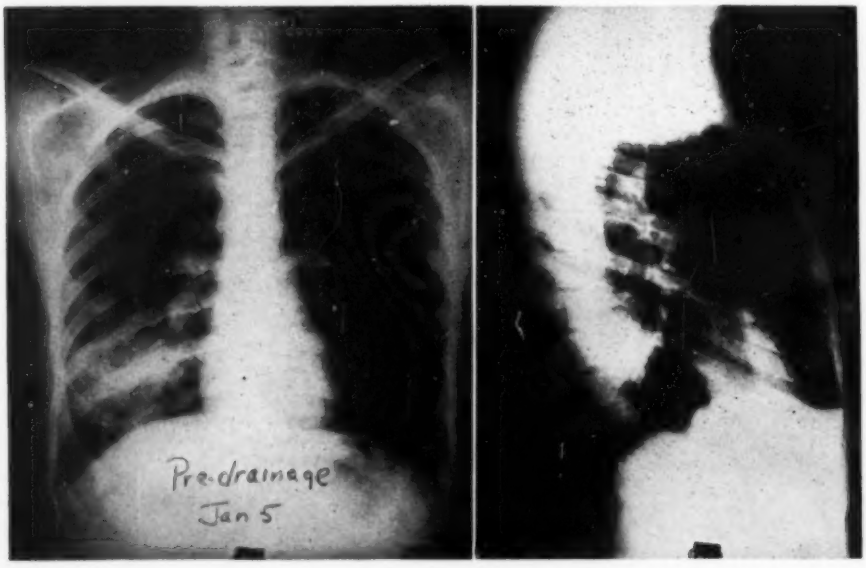

Figure 10, Case 4: Roentgenograms made in the frontal and right lateral projection prior to drainage. There is a large round area of infiltration in the posterior subapical portion of the right lower lobe showing central rarifactions.

age has been attempted and failed resection still can be accomplished, although the hazard is increased somewhat because of the greater chance for intrapleural or wound infections. At operation, the draining sinus should be isolated, excised and inverted with separate instruments, before the chest is opened.

The following case is a good example of failure of cure by external drainage because of secondary changes in the lung. In this case total lobectomy was successful.

Case 4: This twenty three year old Mexican male was referred by Dr. Gerald Scarborough. The patient originally was admitted to the hospital because of a six months' history of intermittent hemoptyses and productive cough which followed tonsillectomy. The initial roentgenogram showed a rounded area in the right lower lobe posteriorly with a central area of necrosis (Fig. 10). Preliminary rib resection was done but a free pleural space was encountered. Later, further rib resection was performed and the cavity entered. A large amount of necrotic debris was removed and the cavity packed. The patient, however, coughed up blood repeatedly following the drainage procedure and bronchoscopy on two occasions showed blood clot in the right lower lobar bronchus. The abscess cavity appeared to be relatively clean but because of the hemorrhages further surgery did not appear to be warranted and the external sinus was allowed to close. Serial roentgenograms showed persisting patchy infiltration in the right lower lobe (Fig. 11). Five months follow-

Figure 11, Case 4: Frontal roentgen film made just prior to lobectomy. The infiltration persists and seems to involve the right lower lobe somewhat more diffusely.

ing the original cavity drainage, a one-stage lobectomy by individual ligation technique was performed. The patient became ambulatory in five days and was discharged from the hospital in about three weeks. The operative specimen is shown in Figure 12.

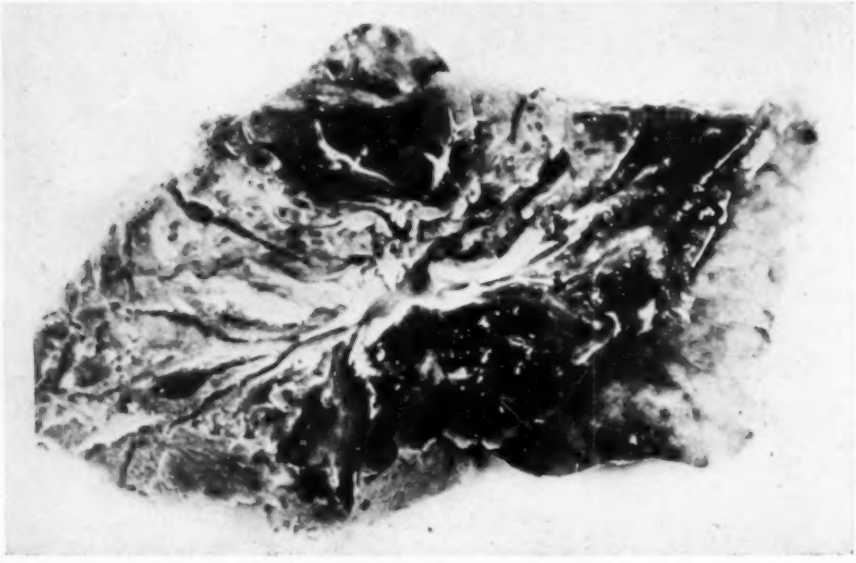

Figure 12, Case 4: The right lower lobe has been sectioned through the long axis of the major bronchi. The diaphragmatic surface is to the right. Extensive bronchiectasis and pneumonitis are present. These findings readily explain why external drainage of the abscess was unsuccessful. The partially collapsed abscess cavity with overlying pleural adhesions can be seen at the left.

SUMMARY

1) The surgical aspects of acute and chronic pulmonary abscess have been discussed.

2) It has been emphasized that pulmonary abscess is a serious and unpredictable disease with ever-present surgical potentialities. It follows that early and frequent consultations between the physician and his thoracic surgical consultant are of the utmost importance.

3) It has been stressed that the arbitrary division of treatment of pulmonary abscess into medical and surgical aspects based on the length of illness is hazardous and has been largely responsible for the appalling mortality rates of past years.

4) The important role of bronchoscopy in improving "internal drainage" has been described.

5) The general indications for surgical drainage in pulmonary abscess have been given.

6) The pathological changes accompanying chronic pulmonary abscess have been briefly presented.

7) The indications for pulmonary resection (segmental lobec-

tomy, total lobectomy, total pneumonectomy) in the treatment of both drained and undrained chronic pulmonary abscesses have been discussed.

8) Case histories have been presented which emphasize the salient facts.

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RESUMEN

1) Se han discutido los aspectos quirúrgicos de abscesos pulmonares agudos y crónicos.

2) Se ha recalcado que el absceso pulmonar es una enfermedad grave e imposible de pronosticar, que siempre posee potencialidades quirúrgicas. Por esta razón consultas tempranas y frecuentes entre el médico y su cirujano torácico son de suma importancia.

3) Se ha recalcado que la división arbitraria del tratamiento de abscesos pulmonares en aspectos médicos y quirúrgicos, basada en la duración de la enfermedad, es peligrosa y ha sido responsable en gran parte por la mortalidad alarmante de los años pasados.

4) Se ha descrito el importante papel de la broncoscopia en mejorar la "canalización interna."

5) Se han dado las indicaciones generales para la canalización quirúrgica del absceso pulmonar.

6) Se han presentado brevemente los cambios patológicos que acompañan el absceso pulmonar crónico.

7) Se ha discutido las indicaciones para la resección pulmonar (lobectomía segmentaria, lobectomía total, neumonectomía total) en el tratamiento de abscesos pulmonares crónicos, tanto canalizados como no canalizados.

8) Se han presentado historiales de casos que recalcan los hechos sobresalientes.

Bronchiectasis*

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Before the use of iodized oils, bronchiectasis was often not recognized. Numerous cases were incorrectly diagnosed as tuberculosis and unresolved pneumonia. At the present time a positive diagnosis can be made in almost every case. Bronchiectasis is an involvement of the lungs; the process may involve only a small portion of one lung, or in advanced cases may affect nearly all lobes.

There are two forms of bronchiectasis: congenital and the acquired. In the first type, deformities develop in fetal life by fibrosing bands and cysts and failure of expansion of the alveoli. The acquired form usually follows pneumonias associated with measles and whooping cough, and any condition which produces an atelectasis of an area supplied by a small or large bronchus. The frequent causes of atelectasis are foreign bodies in the lung, tumors of the bronchi, abscesses of the lungs, and extrinsic factors such as deformities of the chest wall, fluid in the chest, and tuberculosis. Cysts of the lungs, whether congenital or acquired, frequently precede the acquired form of bronchiectasis.

Congenital bronchiectasis is much more common in infants and children, but it is frequently overlooked until complications such as pneumonias occur. The acquired form of bronchiectasis in children is usually due to the aspiration of foreign bodies such as peanuts (arachidic pneumonitis). The foreign bodies produce atelectasis beyond the obstruction of the bronchiectasis. This is usually followed by an inflammatory process with or without abscess formation. Fibrosis of that portion of the lung develops in healing, and bronchiectasis frequently occurs. The pneumonias which follow scarlet fever, measles, chicken pox, and whooping cough predispose to bronchiectasis in children.

When bronchiectasis is found in an adult, the condition may have been of the congenital type, unrecognized for years, until acute inflammation occurs in or around the bronchiectatic area. But the usual acquired form follows, as in children, various types of pneumonias, aspiration of foreign bodies, mediastinal distor-

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tions, compression of the lung by fluid in the pleural cavity, thoracic deformities, and bronchial tumors, benign or malignant. Bronchitis, even with large amounts of secretion, rarely result in bronchiectatic dilatation.

Etiology of Bronchiectasis

Allergic bronchitis has been frequently considered as a precursor of bronchiectasis because of the bacterial infection that is engrafted on the allergic reaction. It is, however, a very uncommon cause of this disease. Spirochetal infection may cause a destruction of the mucous membrane, and even bleeding from the bronchi, which may be followed by dilatations. In this form of bronchiectasis one rarely finds the widely dilated bronchi of the true bronchiectasis. Silicosis is not uncommonly associated with bronchiectasis due to the fibrosis which is a secondary process. Nasal sinus disease has received considerable attention as a possible etiological factor. It is well known that there is frequent association of sinus disease and bronchiectasis. It is not likely that sinus disease itself will produce bronchiectasis, but when both conditions coexist it is likely that an acute flare-up of either one will influence the other. Tuberculosis is a common etiological factor, especially in the chronic ulcerative type, but the amount of secretion which develops in this type is considerably less.

David T. Smith[1] suggested that the presence of fusiform bacilli and spirochetes in the bronchi may be an etiological factor in bronchiectasis. In some of his experimental work he was able to produce bronchiectasis in animals by the injections into the bronchial tree of the above organisms which he had cultured. Most men interested in this field of pathology feel that these organisms are secondary invaders.

Lipoid pneumonia is one of the less common factors in the development of bronchiectasis. This condition is more common in infants or older people who have aspirated oil into their lungs. The aspiration of mineral oil is the usual cause for oil pneumonitis; however in infants, aspiration of milk or cod liver oil may also produce this disease.

Tumors of the bronchi, benign or malignant, may be followed by secondary inflammation of the pulmonary tissue, destruction of the bronchial walls, and bronchial dilatations.

Pathology

In congenital bronchiectasis there is a more or less orderly arrangement of the columnar epithelium, cartilage plates, and mucous glands. Evidence of inflammatory changes are either absent or slight. Multiple, irregular lung cysts may be present; they are

distributed throughout the lung or may be localized in a single lobe due to the compression of the main bronchus, possibly by the duct of Cuvier.[2]

Cylindrical enlargements of the bronchi with atelectasis of the surrounding lung tissue is the usual type of involvement in congenital bronchiectasis. When abscess formation develops, due to pneumonic infection, one finds the usual pathologic appearance of the secondary process in addition to the primary lesion.

In the acute bronchial inflammation, changes usually occur in the bronchial walls. The infection may begin in the mucosa, followed by excretion of considerable mucus and pus. Aspiration of a foreign body may be followed by an infection of the bronchial tree, and fibrous strictures may occur, producing obstruction of bronchi with the resulting dilatation of the bronchial wall. Ulcerations of the bronchial wall and severe hemorrhages may occur if a large blood vessel is eroded. Considerable metaplasia may develop even to keratinization. Because of the strictures and destructions of the bronchial tree above the dilated portions of the bronchial tubes, putrifactive organisms have a suitable soil, and eventually may set up abscess and even gangrene of the lung. It is not uncommon that a broncholith may occlude a large bronchus, following which bronchial infection develops, abscess formation results, and a bronchiectatic area eventually develops. Pleural fibrosis develops in bronchiectasis only when there is secondary pneumonitis.

An acute form of bronchiectasis has been described recently by Brian Blades,[3] particularly in the atypical forms of pneumonia. He found that the dilatations of the bronchi in the lower lobes disappeared after several weeks. The diagnosis of this condition was made after the injection of iodized oils into the bronchial tree. It appears that this type of bronchiectasis could not be due to the pathologic process described above, as the lesions could hardly disappear in so short a time. It is conceivable that there were relaxations of the bronchial walls due to an inflammatory process which later subsided, permitting the bronchi to dilate and contract normally (Figs. 1 and 2).

Clinical Symptoms

Bronchiectasis may be present in two or more members of the same family. It is not uncommon in patients who have so-called "situs inversus." It is important to obtain a good history to determine whether the patient had suffered from pneumonia following chicken pox or whooping cough in childhood, or whether the patient had aspirated a foreign body at any time.

Cough is the most important and persistent symptom. It may

be a slight hacking cough with only slight expectoration, or violent paroxysms with large amounts of sputum, with or without a foul odor. The history usually shows that the cough has persisted for years, and frequently from childhood. The amount of sputum varies from a few cubic centimeters to as many as 1500 cc. a day, and occasionally is streaked with blood. Hemorrhages are more frequent in the advanced cases than in tuberculosis, and occasionally fatal hemoptysis occurs. The odor of sputum at times is so vile that the patient becomes an outcast. In rare instances, bronchiectasis may be present without cough or symptoms, but

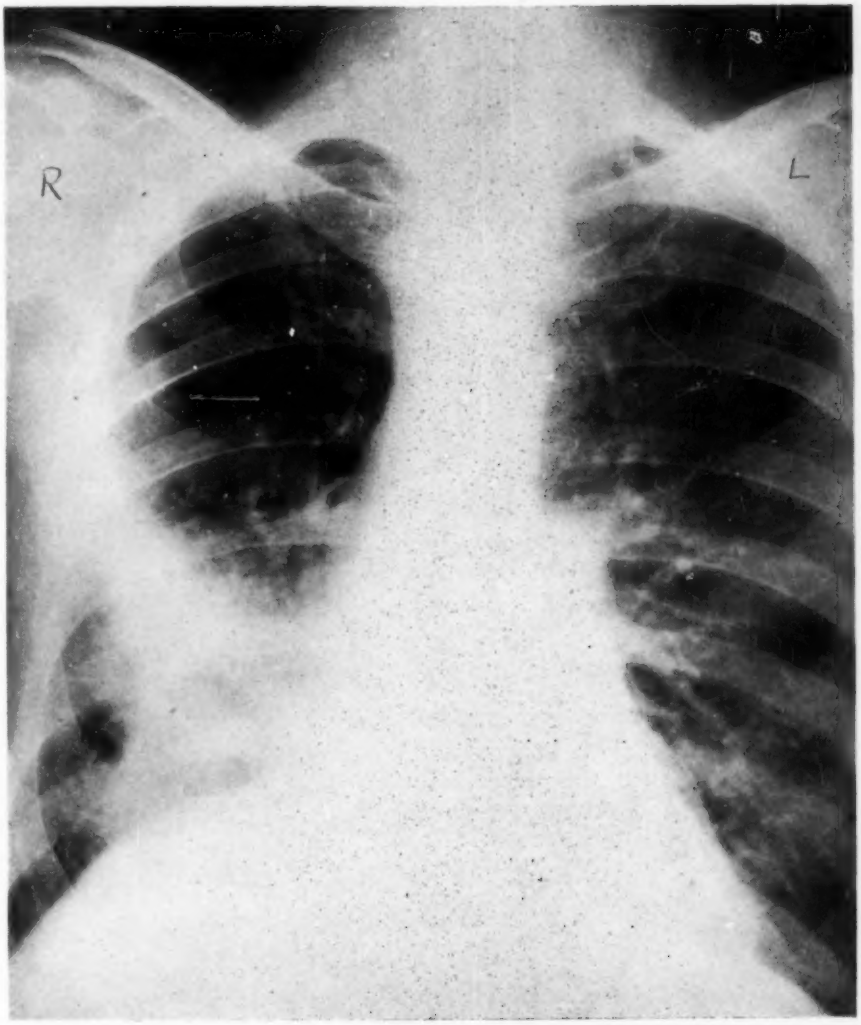

Figure 1: Roentgen film of patient with lipoid pneumonia. In the left cardiophrenic angle one can see a bronchiectatic atelectatic lobe. In the right base there is a diffuse haziness which proved to be pneumonic consolidation and fibrosis (See Figure 2).

lung hemorrhages occur. Studies with iodized oil and roentgen films show the bronchiectatic dilatations, typical of the disease. This has been called the dry bronchiectasis by Bezancon and his associates.[4] This particular type of disease has been frequently mistaken for tuberculosis, but the absence of tubercle bacilli in smears, cultures and guinea pig innoculations rule out this disease.

Bronchiectatic patients often experience fever, sweats, and chills. These episodes frequently occur with attacks of pneumonitis which develops as a result of the spread of the infection from the bronchial tubes through the lymphatic channels to contiguous lung tissue. Pain in the chest is unusual unless there is pneumonia or pleurisy. Constitutional symptoms may be slight for long periods of time unless acute "colds" or acute sinusitis develops. In long-

Figure 2: Photograph of left lung on sagittal section. Left upper lobe is emphysematous; left lower lobe shows bronchiectatic atelectasis. Note the dilated bronchi in this area.

standing cases dyspnea is not an unusual symptom, due to emphysema and cor pulmonale. Osteoarthropathy is a common accompaniment to bronchiectasis.

Physical Signs

In bronchiectasis the physical signs depend on the amount and location of the involvement. In the bronchiectatic, atelectatic cases, compensatory emphysema develops, and surrounds the affected lobe. This masks the signs that can be elicited by auscultation and percussion.

In other cases, impaired resonance is found over the diseased area. Rales are heard, depending on the size of the involved bronchi and the amount of secretion in the tubes. In patients who have large amounts of sputum retained in the bronchial dilatations, diminished breath sounds are noted; but when the patient is given postural drainage or has a violent cough with much expectoration, bronchial breath sounds are heard. The whisper sounds at this time are usually markedly increased, due to consolidation of lung tissue. At other times, practically normal breath sounds and percussion notes are elicited. Roentgen ray studies, both by flouroscopy and films are the best means for the demonstration of the true character of the lesion.

Roentgen Ray Findings: Fluoroscopy and Films

In the fluoroscopic study of bronchiectasis, it is possible to inspect the shadows of the lung, normal and abnormal, the position of the heart, mediastinum, and dipharagm. One can visualize the varying degrees of air-containing lung, and also note emphysematous areas (hyper-aeration). It is important to view the patients from various angles, and it is possible to throw the cardiac shadow away from densities lying in the same planes. By this means, one can frequently see triangular shadows of bronchiectatic, atelectatic areas which are hidden in the posterior anterior position. One can also note the movements of the diaphragm and the various densities which appear as a result of the inflammatory process. The x-ray film makes a permanent record of changes which have been

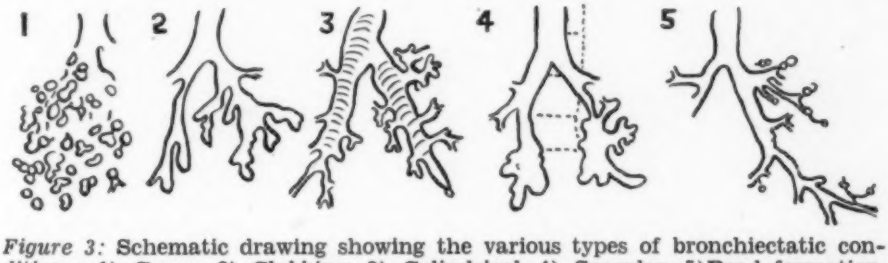

Figure 3: Schematic drawing showing the various types of bronchiectatic conditions: 1) Grape, 2) Clubbing, 3) Cylindrical, 4) Saccular, 5)Bead formation.

found on fluoroscopic study, and it is advisable not only to take stereoscopic films, but also tangential and true lateral. Even with these films, bronchiectatic areas may not be visible without the use of iodized oil instillations into the bronchial tree (Figs. 3 and 4).

Iodized Oil Injections into the Bronchial Tree

There are several methods of introducing iodized oil (40 per cent). The simplest one is the injection into the pharynx of 20 cc. of warmed iodized oil while the patient's tongue is pulled out as far as possible. In many instances, this can be done without the

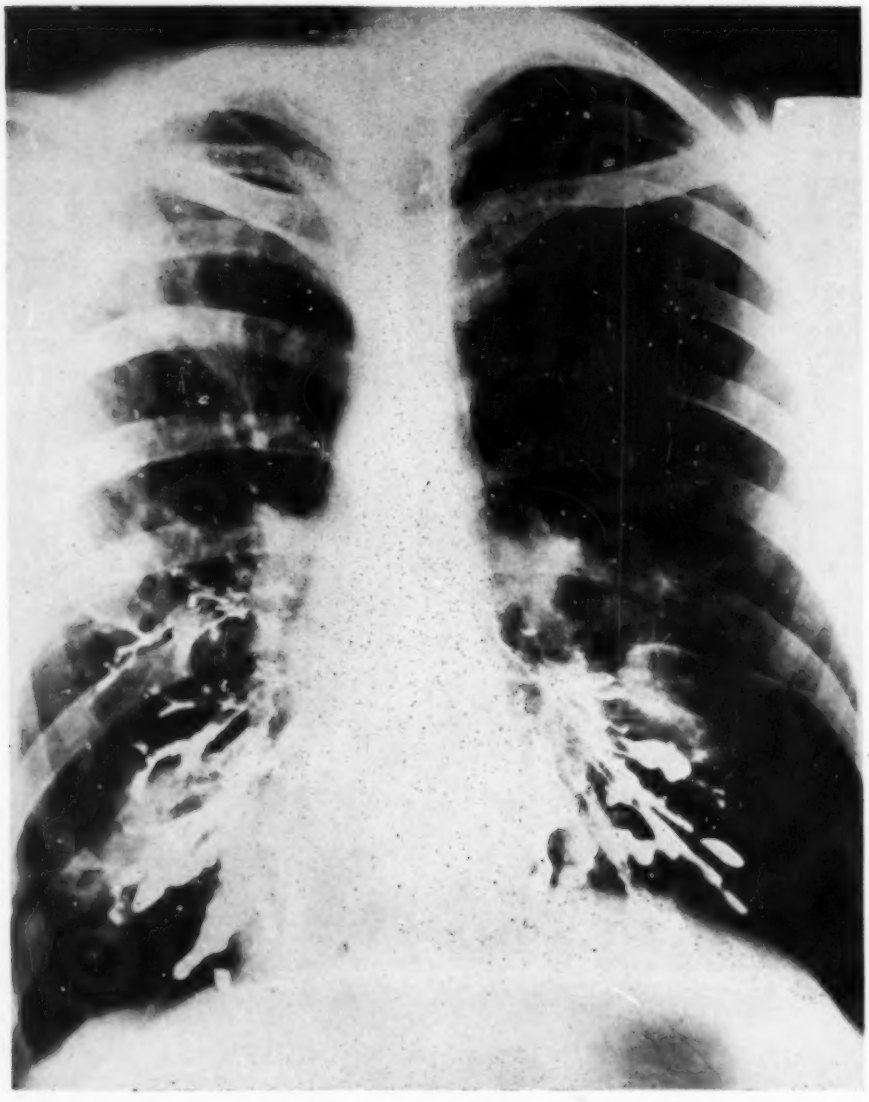

Figure 4: Bilateral bronchiectasis showing various types of dilatations shown in Figure 3. (Iodized oil injection).

use of local anesthesia. When a patient has a sensitive pharynx, it is advisable, preliminary to the injection, to use 10 per cent cocaine solution as a local anesthetic (Fig. 5).

Some of the other methods which are in use are the following: Injecting the oil through a tracheal catheter after the use of a local anesthetic; through a nasal catheter; and through a tube introduced through a bronchoscope directly into the desired bronchus. The author has found that lipiodol (Lafay), which contains 40 per cent by weight of iodine combined with poppy seed oil, has been the most effective.

Not all the iodized oil is expectorated, and may be retained in the alveolar structures for years. It is rare for complications to arise, but patients sensitive to iodine may develop iodism (nasal discharge, congestion of the conjunctiva, and even slight fever and headache). These symptoms disappear within a few days. On rare instances, the oil has been found to block a bronchus and produce atelectasis of a portion of the lung.

When the sputum is emptied into a specimen glass, it separates into three layers. Elastic fibers are rare unless there is a marked degeneration of lung tissue. A microscopic examination shows degenerated, blocked cells, crystals, and fatty acids; lymphocytes

Figure 5: (A) Simple method of introducing iodized oil into the bronchial tree. Straight tip of cannula is positioned midway between the uvula and the tonsil. The tongue is extended as far as possible while the patient is breathing deeply. (B) Iodized oil shown in the interarytenoid fossa and overflowing into the bronchial tree when the vocal cords are separated.

do not predominate. Yeast cells are not an uncommon contaminant. The various micro-organisms, such as staphylococcus aureus, micrococcus catarrhalis, B. mucosus-capsulatus, hemolytic streptococcus, pneumococcus, streptococcus viridans, and fusiform bacillus are the most common bacteria found in the various diseases associated with and predisposing to bronchiectasis. Numerous studies of the sputum for acid fast bacilli should be made, both by concentration method, guinea pig innoculation and culture, to determine whether tuberculosis is present.

The sedimentation test is usually rapid when acute exacerbations take place. This test is not as valuable as in tuberculosis, but is an indicator of the virulence of the bacteria present.

Treatment of Bronchiectasis

Until recent years, the treatment of bronchiectasis has been palliative. Before definite diagnostic procedures were available, the majority of persons afflicted with bronchiectasis were treated as tuberculous patients in sanatoria. It is now recognized that the surgical removal of the diseased lobe or lobes, when feasible, is the only effective cure for bronchiectasis. Unfortunately, many patients present themselves too late for operative procedures. There are benefits, however, to be obtained from medical treatments, particularly: postural drainage, heliotherapy, intravenous therapy, direct intrabronchial application of drugs, inhalation of vapors, bronchoscopy, and abdominal belts. Of lesser importance is the question of rest, diet, and climate. The benefits derived from the above treatments are an amelioration of cough, lessening the amount of sputum, and decrease in the frequency of attacks of pneumonia.

Postural Drainage: In the advanced cases of bronchiectasis, most patients have found by practical experience the best positions to assume in emptying their lungs, and it is not unusual for patients to bring up from 500 to 1000 cc. at one time. This copius discharge relieves the patient of considerable cough which is necessary to bring up sputum, and in that way, they save much wear and tear.

The methods of postural drainage are simple. The patient may be placed on a chair with his head down and his hands on the floor; or be placed on a teeter-totter used by children, where he can be inclined at any angle for a few minutes at a time. For very sick patients, a postural drainage bed has been devised by the author. Without any effort on the part of the patient, the bed may be placed at any incline by moving certain levers and ratchets on the bed.[5] A drainage should be done two or three times a day, preferably in the early morning and before retiring. The

symptomatic relief which frequently follows postural drainage is dramatic, and patients may go about their business or pleasure for long periods of time without any great discomfort (Fig. 6).

Heliotherapy: Heliotherapy has not been effective in reducing the amount of secretion. Patients may feel better generally by exposure to sunlight in moderation. The artificial sunlamps are sometimes beneficial. The application of heliotherapy should be started a few minutes at a time and gradually increased, so that the patient may be exposed for several hours. It has never been quite fully understood how results may be obtained, but clinically it has been found that some patients do improve.

Intravenous therapy — neoarsphenamine: This drug has been used in the same way as in the treatment of syphilis, and occasionally one sees a lessening of symptoms, particularly in the reduction of the amount of sputum and the disappearance of its foul odor. It may be used in patients who show the spirochetes and fusiform bacilli in the sputum. Postural drainage should always be used in conjunction with this therapy.

Intrabronchial application of drugs: Iodized oils and brominized oils (in those patients who are especially sensitive to iodine) may be introduced directly into the bronchial tree. The value of these oils is not in their iodine or bromine content, but rather in the displacement of the purulent secretion. It also mechanically coats the mucous membranes of the bronchial tree and thus prevents irritation of the mucous lining. One of its effects is to dilute the

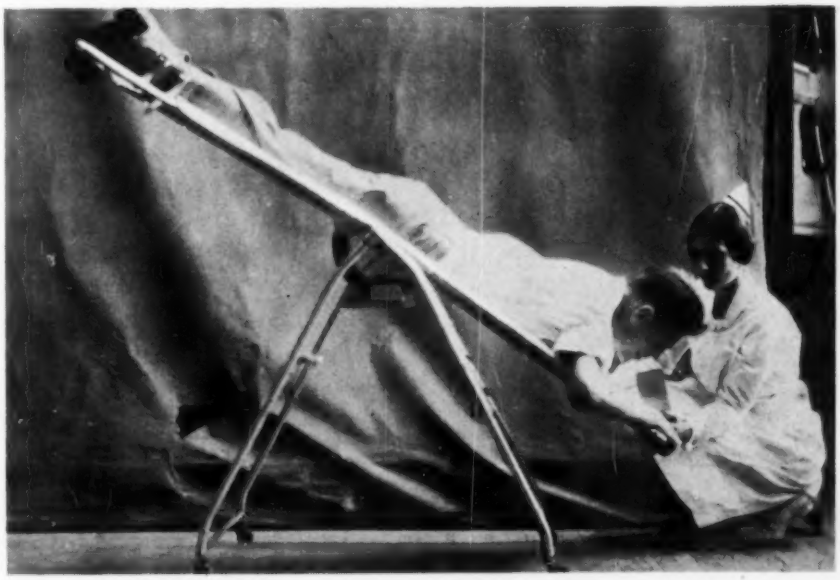

Figure 6: Postural drainage table. Patient can be adjusted to the most suitable position for emptying the bronchi.

secretion so that it may be coughed up easier. Ten to twenty cubic centimeters of oil may be injected once a week, and it should be warmed to body temperature before it is introduced. This treatment should not be kept up for more than four weeks if the results obtained do not warrant it. The methods of instillation of the oil have been described previously.

Inhalations of medicated air: Until 1941 the use of medicated air and volatile drugs had been of only the slightest value in the treatment of bronchiectasis. At that time M. R. Castex[6] suggested the use of a nebulized preparation of sulfathiazole in the bronchial tree. He reported good results in the treatment of suppurative conditions of the lung. Several other authors, particularly Stacey,[7] Harris,[8] and Edlin,[9] have written on this subject. Sulfathiozole (5 per cent) in a normal saline solution is used as a spray. This is given daily over long periods of time. A lessening of the purulent discharge was noted, but the pathologic status of the involved lung remained.

A renewed interest has developed in the use of aerosol with penicillin since the published work of Bryson, Sansome, and Laskin[10] in 1944. These authors found that penicillin aerosol penetrated the lungs, diffused into the blood stream, and was excreted in the urine. Barach[11] and his co-workers suggested the use of penicillin in a normal saline solution to be used as the vehicle for penicillin inhalation. Vermilye[12] used Barach's method of treatment on 200 patients suffering from upper respiratory conditions, and reported excellent results. Nebulization of the penicillin was obtained with the use of an oxygen tank attached to a Vaponephrin nebulizer. "A Y tube is inserted at some point in the rubber tubing, one arm of the Y being left free. The aperture at the top may be attached to the original nebulizer, at the carburetor; this permits some economy in the use of penicillin, but is not essential."

Vermilye found that the calcium salt of penicillin makes a superior aerosol, is less likely to cause cough, and is more agreeable because it has less odor. The dose most used for adults was 25,000 units in 1 cc., but larger doses may be used. From three to five treatments daily can be given to ambulatory patients, and for those too ill the large oxygen tank with the spray at the bedside is used.

It has been recommended by Segal and Ryder[13] that bronchiectatic or lung abscess patients be given a preliminary treatment for at least a week of several inhalations of penicillin per day before operation. Olsen[14] has also used this type of preparatory treatment for lobectomies and has used as high as 75,000 units of penicillin per day. Streptomycin is now being tested in a similar

manner. Sturner[15] suggested that a small amount of epinephrine added to the penicillin mixture would shrink the mucous membranes and thus open the passages for the distribution of the penicillin aerosol and also to prolong its action.

Barach[11] and his co-workers reported that the absorption of penicillin into the blood and its recovery from the urine is not necessarily a measure of the effectiveness of the drug. It is their opinion that penicillin spray exerts its bactericidal effect by contact with the organisms.

Penicillin has been used intratracheally by Kay and Meade, Jr.[16] They used from three to five cubic centimeters of saline solution of the sodium penicillin up to 10,000 units per cubic centimeter. They later increased the strength to 50,000 units daily at one application. The blood penicillin is reported as high as 0.03 Oxford units. In a series of 45 patients with bronchiectasis they found clinical improvement after four to five days, but in the minimal diseases obtained almost complete relief of symptoms. In a series of 50 lobectomies for bronchiectasis in which penicillin was used, as compared with 25 lobectomies in which this drug was not used, no difference was noted in the pathologic study of these specimens. However, these authors concluded that those patients who had had the penicillin treatment before lobectomy recovered more promptly and did have fewer complications.

Romansky, Dugan, and Rittman[17] described still another method for the introduction of penicillin into the lung. They used a preparation of calcium penicillin blended with iodized oil which was introduced directly into the bronchial tree. Every cubic centimeter contained 1500 Oxford units of penicillin. The fluoroscopic picture showed the exact location of the injected oil.

Bronchoscopy: Bronchoscopic drainage of secretions from the bronchial tree has been practiced by a great many physicians. Its value for diagnosis is not disputed, but no eventual cure has ever been described. It is possible by the use of the bronchoscope to dilate strictured bronchi and remove granulation tissue, thus permitting better drainage. Bronchoscopy is not a pleasant procedure, but patients adapt themselves and will permit bronchoscopic drainage because of the great relief frequently obtained. If benefits are not derived within the first few months, it is not advisable to continue with this form of treatment. Along with bronchoscopy, simple postural drainage should be used two to three times daily.

Abdominal belts: Abdominal belts with a compression device to keep the diaphragm high have been of some slight benefit to patients with lax abdominal walls. The cough is more effective and the purulent secretions are expectorated easily.

Surgery of Bronchiectasis

The surgical approach to the treatment of bronchiectasis is firmly established. The mortality rate which was somewhere between 50 and 60 per cent previously is now about 3 per cent. Patients who are submitted to surgical treatments should be in good general condition, preferably under 40 years of age; there should be no evidence of amyloidosis and not too much involvement of both lungs. The present surgical approaches to the treatment of bronchiectasis are by pneumonotomy, lobectomy, and total pneumonectomy.

Pneumonotomy: Pneumonotomy can be used when there are multiple lung abscesses as a complication of bronchiectasis, and where adhesions are so firmly established that it would be too dangerous to attempt pneumonectomy. The operation can be done either with the electric cautery or the actual cautery.

Lobectomy: Lobectomy is now the operation of choice. In the hands of skilled operators it has a low mortality. The original operation as described by Shenstone[18] gave good results. However, with the individual ligation of each vessel at the hilus, as suggested by Kent and Blades,[19] the complications and mortality rates have been even more decreased. It is important that not only the diseased lobe be removed, but the lingula, when affected, must be likewise taken off. Many patients who had been doomed to a life of misery and who were outcasts have been rehabilitated and have become useful members of society.[20] It is unfortunate that so many patients present themselves too late in the disease for successful lobectomies.

Total pneumonectomy:[21] The removal of the entire lung when disease is far advanced is now feasible with the newer techniques devised for pneumonectomy are used. This operation can only be done by skilled thoracic surgeons who are now available in our large medical centers.

SUMMARY

It is evident that until recent times bronchiectasis was frequently diagnosed as pneumonia, tuberculosis, carcinoma, fungus disease of the lung, abscesses, or fibrosis from many causes. But with the methods for definite diagnosis now available, the true character of bronchiectasis is recognized early and suitable treatment may effect cures in a large percentage of patients. The most important conditions giving rise to bronchiectasis are the pneumonias following exanthematous diseases, aspiration of foreign bodies into the lung, and the presence of tumors or cysts which block bronchi, producing atelectasis.

The medical treatments are at best palliative, but sometimes patients may feel clinically well although the pathologic condition remains. Surgery offers positive cures, particularly for young patients. Thoracic surgeons are now available in all medical centers. At the present time, the mortality rate is low, and one should not hesitate to have a lobectomy or pneumonectomy done in suitable cases. The best type of clinic for the study and treatment of bronchiectasis is a chest service, where a chest physician, bronchoscopist, roentgenologist, and thoracic surgeon work as a team.

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RESUMEN

Es evidente que hasta tiempos recientes se diagnosticaba frecuentemente la bronquiectasia como neumonía, tuberculosis, carcinoma, enfermedad fúngica del pulmón, abscesos o fibrosis debida a muchas causas. Pero con los métodos que ahora existen para el diagnóstico bien definido, se reconoce tempranamente el verdadero carácter de la bronquiectasia, y el tratamiento apropiado puede efectuar curaciones en un alto porcentaje de pacientes. Los estados más importantes que producen bronquiectasia son las neumonías que siguen a las enfermedades exantemáticas, la aspiración en el pulmón de cuerpos extraños y la presencia de tumores o quistes que obstruyen los bronquios y producen atelectasia.

Los tratamientos médicos son a lo más paliativos, pero los pacientes pueden a veces sentirse clínicamente bien, aunque persista la condición patológica. La cirugía ofrece curaciones positivas, particularmente a los pacientes jóvenes. Hay actualmente cirujanos de tórax en todos los centros médicos. Al presente la mortalidad es baja, y uno no debe vacilar en someter casos apropiados a la lobectomía o neumonectomía. El mejor tipo de clínica para el estudio y tratamiento de la bronquiectasia es un servicio de tórax donde el médico del pecho, el broncoscopista, el roentgenólogo y el cirujano de tórax, cooperan mútuamente.

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Calcium Ribonate and Vitamin C (Nu 240-10) in the Treatment of Tuberculosis

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The object of this paper is to show that the use of calcium ribonate and vitamin C in the treatment of tuberculosis is definitely indicated. The total serum calcium concentration varies normally from 9 to 11 mg. per 100 cc. It is important to note that the absorption of calcium is governed by these factors: 1) The hydrogen-ion concentration within the intestines. 2) The relative proportion of other substances in the diet. 3) Vitamin D.

Calcium metabolism is easily altered in febrile tuberculous patients because of poor nutrition, of deficiency in free hydrochloric acid, of depressed digestive processes, of reduced alkalinity of the blood and of increased elimination of calcium on account of exudative inflammatory processes.

It is a known fact that in all exudative lesions there is an increase of H-ion concentration in the affected tissues with a relative decrease of calcium. It has also been shown that patients with exudative lesions eliminate more calcium than is ordinarily eliminated under normal conditions. As the result of inflammation, the capillaries become dilated and more permeable, hence serum and its constituents pass into the paravascular spaces. In acute inflammatory conditions this exudate is readily re-absorbed and the tissues involved are restored to their normal state. In chronic inflammation however, some of the exudate remains stagnant and the blood cells found there are eventually disintegrated.

In the presence and action of calcium, the exudate of the paravascular spaces is converted into two histological elements, 1) the matrix, and 2) the fibrils. Eventually these fibrils unite through a process of fusion and later form white waxy bundles of non-elastic fibres. Without the action of calcium the formation of fibrils and fibrous tissue would not take place any more than the

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coagulation of blood in the absence of calcium. Therefore, calcium deficiency is to be considered as a serious handicap for tuberculous patients, inasmuch as it plays such an important part in the formation of fibrous tissue, the forerunner to the healing of some tuberculous lesions.

Since calcification of a pulmonary lesion is considered to be an indication that the process is healed, numerous attempts have been made to promote calcification of these lesions by some form of calcium therapy. Gordon and Cantarow[1] administered parathormone, 20 units every 48 hours, and calcium lactate, 30 grains three times daily, to 14 patients with pulmonary tuberculosis for periods of one to six months. Roentgenographic studies failed to reveal any change in calcification of the lung fields or bones of the hand. They maintained that calcium may exert a beneficial effect upon certain symptoms. Excessive bronchial secretion may be lessened, with consequently diminished cough; the pain of acute pleurisy may be relieved by intravenous or intramuscular injections of calcium gluconate or by parathormone; serobifrinous pleurisy may be benefited.

Menkin[2] noted that approximately 10 times as many calcified areas were found in rabbits injected with calcium chloride than in the control animals. Spies[3] demonstrated that repeated administration of viosterol caused calcification of tubercles. Scholtz[4] gave intramuscular injections of calcium gluconate every second day to 22 patients with exudative tuberculous lesions. This therapy exerted a marked influence on exudation for in 80 per cent of the patients the quantity of sputum decreased rapidly. In many cases the night sweats were favorably influenced. He concluded that the effect of calcium therapy is not due to a specific influence upon the tuberculous process but rather to its vascular action, diminishing capillary permeability and inhibiting inflammatory exudation.

Many writers have recommended the use of calcium in patients with pulmonary tuberculosis, particularly those receiving pneumothorax and those with intestinal tuberculosis. Pisani and Smejkal[5] reported favorably on the prevention of pleural effusions by the use of calcium.

Calcium is of undeniable value in the treatment of diarrhea in intestinal tuberculosis. Brown and Sampson[6] said it was about the best remedy for this purpose. Cantarow[7] suggested that calcium therapy for the diarrhea of intestinal tuberculosis is beneficial because of its inhibitory action on peristalsis and general dehydrating effect. In any event, it has a tendency to relieve abdominal pain, intestinal bleeding and diarrhea in most cases. Pisani[8] in his treatment of 43 cases of intestinal tuberculosis with

intramuscular injection of calcium gluconate found that 20 were definitely improved. Calcium therapy in the majority of cases can bring about changes which tend to rectify the disturbed physiological activity of the intestines.

According to Pisani, the use of calcium for intestinal tuberculosis is of importance because it restores the normal calcium ion concentration of the tissues, it stimulates phagocytosis, stimulates fibrosis and it counterbalances the existing vagotonia through stimulation of the sympathetic filaments. The importance of an early diagnosis of intestinal tuberculosis cannot be overestimated. We doubt if far advanced tuberculous intestinal lesions are amenable to any treatment.

It is generally agreed that the most important function of vitamin C is control of the intercellular cement substance in connective tissue. In this connection, it is of vital importance to tissues of mesodermal origin such as collagen, vascular endothelium, cartilage, and the matrices of bone. Most authors maintain that there is a deficiency of vitamin C in tuberculous patients. Some have shown a rough parallelism between the severity of the tuberculosis and the degree of vitamin C deficiency. In normal individuals the blood-serum level of ascorbic acid is between 0.7 and 2.5 mg. per 100 cc. Elimination of vitamin C from the diet is followed by a gradual fall in the blood level of the vitamin until it reaches a value as low as 0.2 mg. per 100 cc. The urinary excretion of vitamin C in the normal healthy subject is dependent upon the dietary intake.

Faulkner and Taylor[9] have shown that there is an increased demand for vitamin C in infection. Patients with tuberculosis required more than 200 mg. of ascorbic acid a day to keep the plasma level normal. This is to be compared with 25 to 60 mg. which is considered to be the daily requirement of a normal adult. Kaplan and Zonnis[10] have shown that the vitamin C concentration in the blood can be raised in most patients to normal levels by the oral administration of synthetic vitamin.

Singer and Van Bark[11] report that the ordinary balanced hospital diet in itself is adequate in maintaining a vitamin C equilibrium in a large percentage of tuberculous children studies. Controlled feedings of 50 to 100 mgm. of vitamin C daily were followed by a rise in the ascorbic acid values of blood plasma to normal or high levels. Bumbalo and Jetter[12] have shown a comparison of the excretion of vitamin C between tuberculous and nontuberculous children. In the first place, the average basic level of the urinary excretion of vitamin C in the two groups varied considerably. The tuberculous group consistently excreted less than 10 mg. per 24 hours. On the other hand, the control

groups of healthy children excreted on the average of 30 to 35 mg. per 24 hours. As far as could be ascertained, the vitamin C intake of this latter group was approximately similar to that of the tuberculous group, showing that such a diet in a healthy child is adequate, but in one with a chronic infection such as existed in the tuberculous group, it was insufficient.

In consideration of the above-mentioned factors, we have employed at the Boston Sanatorium a combination of calcium ribonate and vitamin C on a series of 50 cases. The preparation was supplied in ampules of 10 cc. containing a 10 per cent solution of calcium ribonate buffered with sodium ascorbate. This new calcium salt is water-soluble to the extent of approximately 70 per cent. It is ionized slowly, and it is believed that in metabolism the acid radical is probably oxidized. Maddock[13] has shown that the intravenous administration of this product on parathyroidectomized and normal dogs, possesses a more prolonged hypercalcemic effect than calcium gluconate. All of our patients received 10 cc. of calcium ribonate intravenously, three times weekly for a period of three months (Table I).

Intestinal Tuberculosis

Our first group consisted of 11 patients, ranging from 18 to 56 years of age, with intestinal tuberculosis. Eight of the patients were females and three were males. The main symptoms presented by this group were diarrhea, abdominal pains, dyspepsia, occasional vomiting, loss of appetite and weight. Four patients failed to respond to ultraviolet therapy. We were able to note a definite improvement in five patients after three months' treatment with calcium ribonate. There was no change in one case and there were five deaths.

TABLE I

The Results of Calcium Ribonate Therapy in Tuberculosis

Cases	No. of Patients	RESULTS		
		No Change	Improved	Dead
Intestinal Tuberculosis	11	1	5	5
Pleural Effusion Following Pneumothorax	10	1	9	0
Pre-Pneumothorax	10	No Fluid in 8 Cases		0
Empyema	6	3	2	1
Far Advanced Pulmonary Tuberculosis	13	2	9	2

Case 1: S.C., a 48 year old, white, married female was admitted with a positive sputum and far advanced pulmonary tuberculosis. Roentgenograms of the chest revealed infiltration and moderate broncho-pneumonic reaction involving the entire left lung with a large cavity in the left apical area. The patient was admitted with a two-months' history of cough, expectoration, night sweats, loss of appetite, and 17 lb. in weight. After one week's hospitalization the patient began to complain of abdominal pains, nausea, and diarrhea ranging from 6 to 10 bowel movements a day. Roentgenograms of the gastro-intestinal tract revealed the presence of intestinal tuberculosis. The patient was placed on calcium ribonate therapy and after 6 weeks' treatment her diarrhea, nausea, and abdominal pains had disappeared. At the end of three months' treatment, the patient had gained 22 lbs. A left thoracoplasty is to be performed in the future.

Pleural Effusion Following Pneumothorax

Our next group consisted of 10 patients ranging from 16 to 42 years of age, who had developed a pleural effusion during the course of their pneumothorax. Six of the patients were females and four were males. The pleural effusion had existed anywhere from 2 months to 9 months. On calcium ribonate therapy, we were gratified to note that the fluid disappeared in 9 cases and there was no change in one case which required thoracentesis.

Case 2: N.B., a 16 year old, single, white school girl, was admitted with a positive sputum and moderately advanced pulmonary tuberculosis. Roentgenograms revealed infiltration and moderate broncho-pneumonic reaction involving the upper half of the right lung. The patient was admitted with a 6 weeks' history of cough, expectoration, loss of appetite and 10 lbs. in weight. A right artificial pneumothorax was instituted and a good collapse was obtained. Three weeks following the initial pneumothorax, the patient developed a right pleural effusion. The patient was placed on calcium ribonate therapy and the pleural effusion completely cleared after 5 weeks treatment. The patient has since been discharged from the sanatorium.

Prepneumothorax

We selected for our next group 10 patients ranging from 18 to 38 years of age, who were to be given pneumothorax therapy. Seven of the patients were females and three were males. Calcium ribonate was administered one month prior to pneumothorax and continued for 2 months during their refills. We noted that only 2 of the 10 patients developed fluid. In a control group of 10 cases, six patients developed fluid.

Case 3: C.C., a 20 year old, white, single student nurse, was admitted with a positive sputum and minimal pulmonary tuberculosis. Roentgenograms revealed infiltration and moderate bronchopneumonic reaction in the region of the 1st right interspace. The patient was admitted with a 7 weeks history of slight cough, fatigue and 5 lb. weight loss. A right pneumothorax was advised. One month prior to receiving pneu-

mothorax, the patient was placed on calcium ribonate therapy and this was continued for two months. She has been receiving right pneumothorax for 16 months with no evidence of fluid in the pleural cavity.

Tuberculous Empyema

Our next group consisted of 6 patients, ranging from 21 to 39 years of age, with tuberculous empyema. Four of the patients were male and two were female. These patients had thoracenteses on the average of every 2 weeks. After three months of calcium ribonate therapy, we noted that there was definite improvement in three patients, in that the fluid became less viscid and the amount of fluid decreased requiring less frequent thoracenteses. No change was seen in two cases and one patient died.

Case 4: T.C., a 21 year old, white, single female was admitted with a positive sputum and moderately advanced pulmonary tuberculosis. Roentgenograms revealed infiltration and small cavity in the right upper lobe. The patient was admitted with a 2 months' history of cough, expectoration, weakness, and loss of 17 lbs. in weight. A right pneumothorax was instituted and after 5 weeks' treatment the patient developed a tuberculous empyema. The patient was tapped on the average of once every 2 weeks for a period of 3 months. On each thoracentesis from 20 to 30 oz. of greenish-yellow fluid was obtained. The patient was placed on calcium ribonate therapy and after 3 months' treatment the fluid practically disappeared. She has not been tapped for 4 months and has since been discharged from the Sanatorium with a negative sputum and partial atelectasis of the right lung.

Far Advanced Pulmonary Tuberculosis

Our next group consisted of 13 patients ranging from 27 to 52 years of age with far advanced pulmonary tuberculosis. Nine of the patients were males and four were females.

The majority of these cases had been in the sanatorium from 6 months to 2 years. None of these patients were considered suitable for collapse therapy. After 3 months treatment on calcium ribonate we found that 9 patients were definately improved. Decrease in night sweats, lessening of cough, and improvement in appetite were especially noted. In 2 patients there was no change and 2 patients died.

Case 5: H.P., a 31 year old, white, married male was admitted with a positive sputum and far advanced pulmonary tuberculosis. Roentgenograms revealed soft infiltration and moderate bronchopneumonic reaction with multiple small cavities involving both upper lobes. The patient was admitted with a 4 months' history of cough, expectoration, weakness, loss of appetite and 12 lbs. in weight. Dut to the patients' clinical condition and extensive disease, no collapse therapy could be undertaken. After 3 months' treatment on calcium ribonate a definite improvement in the patients' cough, strength, and appetite was noted. He gained 20 lbs. in weight. The patient has recently been started on pneumoperitoneum treatments.

No ill effects or untoward symptoms were noted in the intravenous administration of calcium ribonate.

In view of the findings of many workers in tuberculosis and our own experience, we believe that the use of calcium ribonate and vitamin C has a definite place in the treatment of tuberculosis particularly intestinal tuberculosis and pleural effusion.

SUMMARY

1) The writer's observations as well as other investigators' have revealed that there is a definite use for calcium and vitamin C therapy in the treatment of tuberculosis.

2) In a series of 50 cases at the Boston Sanatorium a combination of calcium ribonate and vitamin C was administered intravenously three times weekly for a period of three months.

3) The results obtained justify the continued use of calcium ribonate and vitamin C in the treatment of tuberculosis, especially intestinal tuberculosis and pleural effusions.

4) No ill effects were encountered in the intravenous administration of calcium ribonate in any of our cases.

RESUMEN

1) Las observaciones del autor, lo mismo que las de otros investigadores, han revelado que existe un uso bien definido para la terapia con calcio y vitamina C en el tratamiento de la tuberculosis.

2) En una serie de 50 casos en el Sanatorio de Boston se administró una combinación de ribonato de calcio y vitamina C por la vía intravenosa tres veces a la semana por un período de tres meses.

3) Los resultados obtenidos justifican el uso continuado del ribonato de calcio y vitamina C en el tratamiento de la tuberculosis, especialmente tuberculosis intestinal y derrames pleurales.

4) La administración intravenosa del ribonato de calcio no causó efectos contraproducentes en ninguno de nuestros casos.

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Cystic Disease of the Lung with Report of a Case*

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The problem of cystic disease of the lung has received increased notice in the literature since 1925 when Koontz[1] reported a case and analyzed 108 others, all of which were found in the foreign literature. Wood[2] in 1934 added a series of 16 from the Mayo Clinic. Sellers[3] in 1939 collected well over 400 cases. Recently Edwards and Ehrlich[4] found two cases of lung cyst in a study of about 13,000 inductees and National Guardsmen in New York City which is a fair estimate of the relative infrequency of the condition. In the past eight years six cases of cystic disease were seen at the Department for Diseases of the Chest of Jefferson Hospital, one of which will be discussed in detail.

The term cystic disease has been applied to a wide variety of abnormal localized distensions of the parts of the bronchial tree and pulmonary parenchyma, such as congenital cystic disease of the lung, acquired cystic disease of the lung, cystic bronchiectasis, bulbous emphysema, pleural blebs, and pulmonary pneumatoceles. This diverse terminology has arisen through the efforts to explain its pathogenesis.

There are two schools of thought as to the etiology of cystic disease, one considers it congenital and the other, acquired. The theory of congenital cystic disease is supported by the finding of lung cysts in the fetus and newborn; the frequent association with other congenital abnormalities such as dextrocardia, congenital heart disease and polycystic kidney; and their occurrence in accessory or abnormal lobes of the lung. Some of the supporters of this theory believe that the cyst formations are the result of an arrested development of the bronchial tree while others attribute this pulmonary anomaly to inflammatory processes occurring during intra-uterine life. However, recent reports showing development of cystic disease following infection suggests their acquired nature. It appears likely that both types do occur.

Pathologically, cystic disease can be divided into two groups according to the presence or absence of epithelial lining. The epithelialized can be congenital or acquired. In the congenital, the

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wall contains bronchial elements such as cartilage, glands, smooth muscle fibres and the epithelium is bronchial in type (stratified ciliated). The acquired usually does not have cartilage or muscle in the wall and the epithelization results from the ingrowth of epithelium from the bronchial fistula. The non epithelialized has a wall consisting merely of connective tissue without epithelium, no bronchial elements being present.

Clinically, two main forms of cysts can be distinguished, the solitary and the multiple.[5] Single cysts may be of two types—a large solitary balloon cyst or the small solitary cyst about the size of an orange. This single cyst may lie within the parenchyma of the lung, or it may occupy the site of a whole lobe, or it may arise from other structures to which it is developmentally attached, such as the trachea or esophagus. The multiple form is more common and consists of numerous small cysts representing a more or less diffuse cystic appearance of larger or smaller parts of the lung. They vary greatly as to size, distribution, type of bronchial communication and nature of content. They are often confined to one lung but may be distributed in both.

Numerous theories have been advanced as to the mechanism of producation of lung cysts. Oughterson and Jaffel[6] give a comprehensive review of the subject and conclude that varying degrees of bronchial obstruction plays a major part. The large size of the solitary cyst can be explained on the basis of a check valve.

The clinical picture depends on the type of cystic disease present. The large balloon type of cyst produces symptoms early. It may contain air or fluid or both and can be the site of superimposed infection resulting in pus and surrounding pneumonitis. It is most commonly found in infants and may produce such distension within the thorax that respiratory and circulatory embarrassment results from the shift of the mediastinum and collapse of the displaced lung. It may persist into adult life in an asymptomatic form and be detected accidentally or due to rupture or infection. Smaller solitary cysts are usually silent and only discovered during some respiratory disease necessitating x-ray study. Multiple cysts are usually silent clinically so long as they are sterile and filled with air or mucus. Symptoms are readily produced if infection occurs and the condition may then simulate widespread bronchiectasis or abscess formation, with cough, copious expectoration and dyspnea. Rupture of cysts following exertion has been reported with the symptoms of a spontaneous pneumothorax.[7]

The diagnosis of cystic disease depends mainly on x-ray and bronchography. It should be kept in mind when infants develop progressive respiratory difficulty. In later life the appearance of pulmonary insufficiency in patients without ascertainable cause

should suggest the condition. Cystic disease must be differentiated from the following conditions: spontaneous pneumothorax, emphysematous blebs with valvular bronchial obstruction, pleural blebs, abscess cavities, cystic bronchiectasis and thin walled cavities of other origin.

Treatment depends on the type of cysts and the symptoms they produce. Solitary balloon cysts require excision for there is danger that they may become infected or rupture. Progressive dyspnea due to ballooning out of a cyst or rupture necessitates deflation by aspiration, with subsequent operation. Small isolated cysts require no treatment unless infected. In that case, removal is necessary only if postural drainage and bronchoscopy fail to effect a cure. Lobectomy or pneumonectomy is indicated where extensive cystic change is present in one lung to obviate the danger of infection. Bilateral advanced cases must be treated symptomatically, as in advanced bronchiectasis.

The difficulty in differential diagnosis between spontaneous pneumothorax and lung cyst has been reported by other clinicians.[7,8] The following case is indicative of the problem with a successful result:

REPORT OF CASE

A woman aged 35 was referred because of persistant dyspnea. Past history was irrevelant. No complaints were present until the summer of 1940 when shortness of breath was noticed. Absent breath sounds were detected on the right by her physician when she consulted him during an attack of "grippe" in December 1940. A roentgenogram taken at that time (Fig. 1), revealed what appeared to be a spontaneous pneumothorax on the right and she was placed on absolute bed rest for 3 months. No re-expansion occurred and dyspnea became more marked.

Examination fluroscopically in April 1941 revealed a marked tension pneumothorax on the right, with the lung compressed to the base and the heart and mediastinum shifted to the left. No fluid was present. Adhesions at the costophrenic angle were suspected. Intrapleural readings were 0,+4. The diagnosis of spontaneous pneumothorax with check-value fistula was made and deflation begun. Repeated removal of 2000-3000 cc. of air every 2-3 days over a period of 2 months failed to produce sustained re-expansion of the lung. On occasion difficulty was had in entering the apparently free pleural space and obtaining "good" readings. The reason for this did not become apparent until later. The last aspiration of 3750 cc. of air on May 24, 1941 produced no re-expansion of the lung.

A thoracoscopic examination was then advised to determine whether the suspected adhesions at the base might not be holding open a tear in the lung. This was done by Dr. George Willauer on May 31, 1941. No adhesions were found. Those suspected on x-ray were really the base of a giant cyst which completely filled the pleural cavity and which was attached in many places to the lateral chest wall. The lung was freely expanding though compressed to the base. Numerous puncture wounds on the inner surface of the parietal pleura and the lateral cyst wall

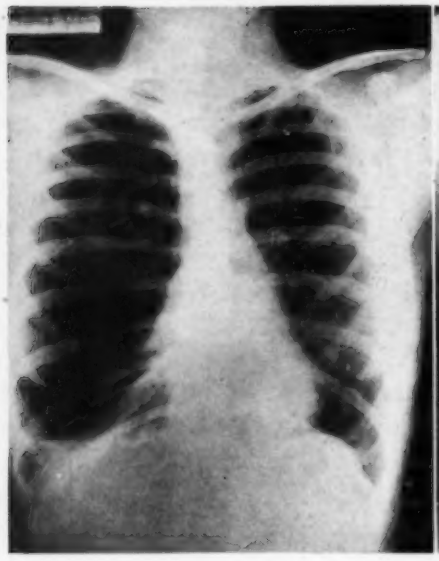

FIGURE 1

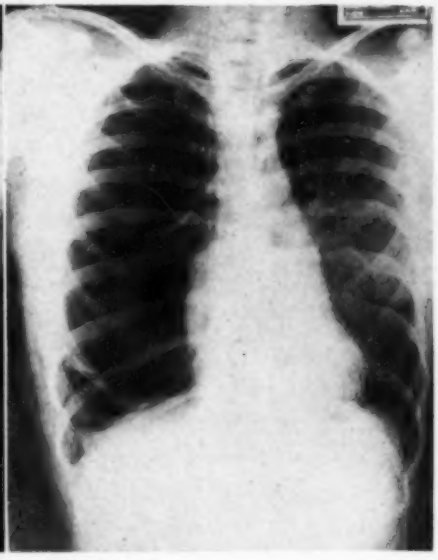

FIGURE 2

Fig. 1: X-ray of December 1940, showing what appears to be a complete pneumothorax on the right with the lung compressed to the base. Adhesions are suspected in the costophrenic angle.—*Fig. 2:* X-ray three days after exploratory thoracoscopy. The walls of the intrapleural cyst are now visible due to escape of air into the pleural space. Cyst is adherent to the lateral chest wall in the lower half of the pleural space.

FIGURE 3

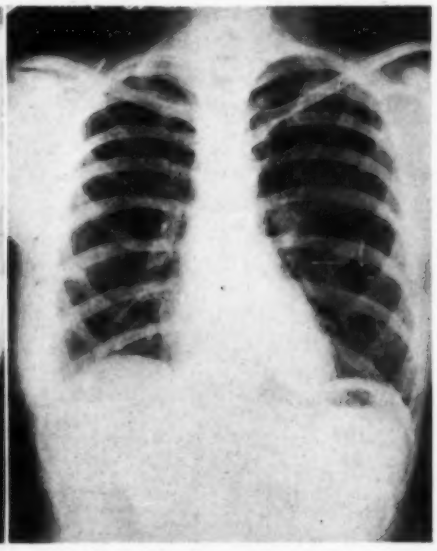

FIGURE 4

Fig. 3: X-ray taken two weeks after cyst was removed, showing 80 per cent re-expansion of the lung.—*Fig. 4:* X-ray ten months after removal of cyst, showing complete re-expansion of the lung and reabsorption of the fluid. Lung fields appear normal at this time.

were visible. Figure 2 shows the x-ray appearance three days later, the walls of the cyst now being clearly defined due to the escape of air into the pleural space following introduction of the thoracoscope.

Three weeks later the cyst was removed. It was found to arise from the apex of the right upper lobe and to be attached diffusely in the pleural cavity. A partial resection of the right upper lobe was done. Effusion developed but was removed by frequent aspirations. Figure 3 taken two weeks postoperatively shows 80 per cent re-expansion of the lung. Complete re-expansion occurred by February 1942 (Fig. 4), with complete absorption of the effusion. Patient is now well and free from complaints.

DISCUSSION

The presence of a cyst was not suspected, despite the failure of sustained attempts at re-expansion on deflation, since it was thought that this was due to a patent broncho-pleural fistula. Repeated fluroscopy and chest films did not reveal the commonly seen trabeculae which would have aroused our suspicion.

Thoracoscopy readily explained the difficulty which was had at times with obtaining "good" intrapleural readings. In those instances the needle was apparently inserted between the parietal pleura and the lateral cyst wall. It was only when the needle entered the cyst itself that "satisfactory" readings were obtained.

Examination of the resected lung revealed that the cyst was in direct communication with a bronchus and at the time of operation this communication was demonstrated by a probe. The cyst wall consisted of fibrous tissue and was lined by a flattened layer of cells. The recent development of dyspnea was probably due to the occurrence of a ball-valve obstruction in the bronchus connecting the cyst to the air passages, allowing air to enter more easily than it could leave it. This was not demonstrated, however, on the removed specimen.

The origin of this cyst was probably congenital in nature, arising in some malformation of the upper lobe and producing no symptoms until adult life.

SUMMARY

1) A brief summary of the etiology, classification, clinical features and treatment of cystic disease of the lung is presented.

2) The difficulty in differential diagnosis between spontaneous pneumothorax and lung cyst is illustrated by report of a case with successful surgical intervention.

RESUMEN

1) Se presenta un breve resumen de la etiología, clasificación, rasgos clínicos y tratamiento de la enfermedad quística del pulmón.

2) Se informa sobre un caso en el que se intervino quirúrgica-

mente con buen éxito, y que sirve de ejemplo sobre la dificultad en el diagnóstico diferencial entre el neumotórax espontáneo y el quiste del pulmón.

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Legislative Implications of Adequate Tuberculosis Control*

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Tuberculosis, a Major Public Health Problem

"Tuberculosis is a community public health problem. It is as sound a public economy to provide the necessary medical and social services for its control and prevention as it is to spend public moneys for a safe water supply, proper sewage disposal, and fire and police protection.

"Tuberculosis is a chronic, relapsing disease and, since one case comes from another, the disease is essentially a slowly spreading epidemic. Tuberculosis is a costly disease; hospital care takes months and even years, and it is expensive. A negligible proportion of patients are financially able to meet the cost of hospitalization—accordingly the taxpayer is paying the bill whether he realized it or not."[1]

The above statement is simply an introduction to the fact that large legislative appropriations are justified and, as will be shown later, are essential requirements for adequate tuberculosis control.

Tuberculosis Laws—Rules and Regulations

Texas laws are basic and, for the most part, can be applied to restrictive measures to prevent the spread of infection, but the laws we have are not enforced. For example: Physicians do not report active cases of tuberculosis that they see, and directors of local boards of health are reluctant to stir up opposition by enforcement of the law since they hold appointive positions. The directors of local boards of health fail to inspect quarters where death from tuberculosis have occurred, and enforce upon owners the sanitary renovations necessary before another family is allowed to move in—due to lack of adequate funds for employment of enough sanitary inspectors. Compulsory measures for the management of individual cases of tuberculosis are rarely required. Most people have an aversion to, and fear of, legal entanglements. Moral persuasion, and holding out to recalcitrant tuberculous persons the existing compulsory regulations, will often be sufficient to gain their cooperation. People cannot be legislated into being

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good but can be influenced by forceful, tactfully trained public health workers and leadership into cooperation and, only occasionally, will it be necessary to apply the basic compulsory laws we already have.

Essentials of Tuberculosis Control

Surgeon General Parran, U. S. Public Health Service,[2] summarizes the essentials of tuberculosis control as follows:

1. X-ray examination of the entire population.
2. Follow-up of every case of tuberculosis discovered by x-ray.
3. Periodic examinations, including x-ray examination, of persons with inactive disease.
4. Prompt treatment for patients with active disease.
5. Strict isolation of open cases to prevent further spread of the disease.
6. Intensified health education activities among the general population, patients, and their families.
7. Expanded research in tuberculosis and control measures.
8. Financial aid to the bread winner and his family.

To carry out a program of such magnitude, it is evident, that an official agency is necessary and, for this phase of the organization, Parran outlines a pattern with the following comment:

"In the National Program, the accepted pattern of Federal assistance to the States would be applied under specific authorization of funds by the Congress. State Health Authorities, in turn, would take the leadership in the *establishment and support* of tuberculosis control programs in local *health departments*. Local health agencies, official and voluntary, would coordinate all available community resources, working closely with the medical profession and welfare groups whose help will be needed to the fullest."

With the above pattern in mind, it is evident that the foundation and superstructure of a Texas State-wide tuberculosis control program should be under the direction of the Tuberculosis Control Division of the Texas Department of Health.

Comparing the Above Program with a Tuberculosis Control Program Proposed by the Board of Directors, Texas Tuberculosis Society

We find conflict of authority interfering with a unified program and a division of responsibility. The six objectives outlined are right in principle, but wrong as to application.

There is revealed a division of responsibility in that the Texas State Department of Health is to take over a part of the control program; that the State Board of Control is to take over the medical policies of hospitalization of tuberculosis cases; and, that the Tuberculosis Committee of the Texas Medical Association shall

offer both departments an Advisory Committee. Wrong! Advisory committees, unsolicited, are usually resented, and are of little value. County and State sanatoria are just as much a part of a tuberculosis control program as is the establishment of other phases of the control program. Either the whole program should be handled by the Board of Control or legislation authorizing the establishment of a Tuberculosis Control Board, composed of a Directorate of Tuberculosis Specialists (three in number), nominated by the Tuberculosis Committee of the State Medical Association of Texas, and two lay members of the Texas Tuberculosis Society, thereby forming a directorate (not simply advisory) with authority to act and, under whom, the Director of the Division of Tuberculosis Control will carry out the details of establishing clinics, employing field nurses, and all other machinery of an efficient program authorized by the Directorate. The Superintendents of State Sanatoria would run their individual institutions in cooperation and in accordance with the Directorate of the Texas Division of Tuberculosis Control. The financial accounting should be set up either through the Texas Board of Control or a state auditor. Upon this point I am not competent to pass. Legislation eliminating the above division of authority and obviating conflict is essential to a unified, well-coordinated program. We talk blithely of a campaign to control tuberculosis and applaud and endorse "The Essentials of a Tuberculosis Control Program," as outlined by Parran, and our Legislature would probably agree to such a program in theory, but when it comes to the appropriation of funds to carry out such a program, they would want to know the approximate cost.

A Yardstick of Measure is Submitted

In December of 1945, while in Washington, New York, and New Jersey, I obtained the following information:

A representative of the U. S. Public Health Service stated that the data they had obtained on the latest, up-to-date, modern hospital construction indicated that the cost of construction of hospitals would be from $6,000 to $7,000 per bed (fully equipped), and the cost of maintenance from $6.00 to $7.00 per day per patient, with a full complement of personnel.

A visit to one of the New York up-state district hospitals at Onconto brought out the fact that the Homer T. Folks District State Hospital cost (ten years ago) approximately $5,500 per bed, and the present cost of maintenance was approximately $6.00 per day, which cost included complete staff and personnel. The construction of this hospital was excellent, consisting of single rooms, two-bed wards, and four-bed wards. Surgical collapse therapy was

done by a rotating team going from one district state hospital to another. Designated members of the staff made diagnostic itineraries to county health units of the counties of the district at stated intervals, taking with them a portable x-ray unit, and held clinics and conferences previously arranged by the field nurse.

I also visited a modern county sanatorium at Broad Acres, Utica, New York. The construction cost of which was around $5,000 per bed, fully equipped, and the maintenance cost of between $5.00 to $5.50 per day per patient. In the State District Sanatorium, one-half of the per-diem cost was charged to the county from which the patient came, and in the county sanatorium, the State paid one-half of the per diem cost of each patient.

In the State of New Jersey, the same rule applies as to sharing the per-diem cost per patient. Hudson County Tuberculosis Sanatorium, a unit of the Medical Center of Jersey City, is a magnificent institution. This institution, completely equipped with private rooms, two-bed rooms, and no ward of more than six beds, cost between $6,000 and $7,000 per bed, and maintenance for same is between $6.00 and $7.00 per day per patient.

Cost of hospital construction today is approximately $6,000 per bed.

When we consider that private hospitals charge from $5.00 to $7.00 per day, exclusive of laboratory and x-ray work, and utilize special nurses—charged to the patients—it is reasonable to assume that $6.00 per day per patient for efficient and adequate service is a reasonable cost with the present-day inflation.

Applying the above Yardstick of Measure:

4,000 additional beds at $6,00 per bed	$24,000,000
4,000 additional beds' maintenance at $6.00 per diem	8,000,000
Financial aid to bread winner: This is figured upon the basis that 1 in 10 would so qualify or 400 at $60.00 per month. (The above fund would best be handled through local welfare agencies rather than through the family to be expended foolishly)	288,000
To the Texas State Department of Health Tuberculosis Division—annually	620,510

The $24,000,000 cost of construction can probably be reduced by Federal aid to $16,000,000. The $16,000,000 can further be reduced by cities and counties who have already voted bond issues. There will also be a further saving in the event Fort Ringgold is utilized

as a District State Sanatorium; nevertheless, a sizable sum will be required for construction to make 4,000 additional beds available.

The $8,000,000 maintenance cost to the State can be reduced one-half if the state and counties share alike the per diem cost of patients hospitalized in state and county hospitals.

The above figures may sound grandiose but to win against the enemy our Armed Forces demanded the best equipment, a full complement of trained personnel, unlimited financial support, and, until such was available, fought defensive battles, but as soon as the above requisites were available, a vigorous aggressive campaign resulted in shortening the time for subduing the enemy, thus saving many lives and money in the end—the same will be true in the fight against tuberculosis.

Supplying 4,000 additional beds is not the only essential of adequate tuberculosis control, but the establishment of pneumothorax and diagnostic clinics in thirteen areas, the employment of clinic and tuberculosis field nurses, part-time and full-time tuberculosis specialists, clerical assistants for tuberculosis registry, purchasing or aiding in the purchase of fluoroscopic equipment, purchasing or aiding in the purchase of x-ray equipment, along with the employment of sufficient personnel, is no small order and, as estimated by me, would be approximately $620,510 the first year. However, from my observation and experience, such a program of early diagnosis, pneumothorax treatment, supervision of cases returned from the sanatoria, and follow-up of all cases showing significant findings is so important that, in the beginning, new hospital construction should be on the basis of one bed to each death and so constructed that additional units may be added as needed; i.e., that 2,000 additional beds be built in localities where the death rate is high, and no units of less than 100 to 150 beds be constructed; that the diagnostic and pneumothorax clinics be instituted without delay with an approximate expenditure through the Tuberculosis Division of the State Department of Health of $620,510.

The item of financial aid to the bread winner and his family, while he is incapacitated, is so obviously an essential as to need no discussion unless it be the minimum or maximum amount per month.

Since 1917, there has been a decrease of 68 per cent in the human death rate from respiratory tuberculosis and 84 per cent in other forms due, to a considerable extent, to the elimination of the disease in cattle."

Stewart,[4] in an article "Tuberculosis Control," made the following comment:

"It is helpful to recall that in spite of violent opposition, approximately 8,000 veterinarians applied tuberculin tests to 25,000,000 cattle in 1925, and applied nearly 250,000,000 tests between 1917 and 1942, inclusive. The reduction of bovine tuberculosis to the vanishing point, accomplished by a relatively small group of veterinarians in the course of 25 years, challenges 150,000 licensed physicians in the United States to eradicate human tuberculosis."

Adequate Appropriations for Tuberculosis Control is Sound Economy

The National Tuberculosis Association estimates that every death from tuberculosis represents an economic loss (in treatment and loss of employment) of $10,000. By the same token, the economic loss from the 2,923 deaths in Texas in 1945 was $29,230,000.

From Canada, Richards[5] reports: "Ontario has a very active, efficient prevention department which has calculated that it costs approximately $500 to find each case of tuberculosis; about $1,000 to treat each minimal case of tuberculosis, $3,000 for each moderately advanced case, and $5,000 for each far advanced case."

Dublin,[6] at an annual meeting of the American Public Health Association, made this statement:

"It is estimated that in two years, 1937-1939, when Detroit City Council was backing the Health Department with $400,000 in the tuberculosis case-finding program, more than $1,300,000 was saved to the City of Detroit in hospital bills by discovering and treating cases in the early stages of the disease and through the prevention, by this means, of additional cases."

These facts should be brought to the attention of the voters of Texas and the legislators by well organized groups throughout the state. In the recent race for governor, one candidate brought out the fact that only eight cents per capita was spent annually for the public health, stating, "that is a disgrace," which it is. In the Framingham Demonstration, a summary of which is submitted, the following statement in conclusion was made:

"The most significant result of this attempt to control tuberculosis was the way in which community sentiment was aroused, so that the total cost of health work from all agencies increased from 40 cents to $2.40 per capita. These figures indicate that lack of funds can be overcome provided proper leadership and education is available."

Whether legislative appropriation of funds by direct taxation on real estate or by sales tax should be made, is open to debate and decision, but the fact that the American people can afford to pay for tuberculosis control is illustrated by the fact that in 1940, the people of the United States spent:[7]

$ 56,721,746 for chewing gum
$134,525,233 for cigarettes
$282,002,617 for ice cream

M. Scarborough, County Judge of El Paso County, made the following comment:

"We have a county hospital that we are unable to maintain to full capacity—and I am of the opinion that regional hospitals established by the State and either maintained by the State or partially maintained by the State with the balance furnished by counties is the solution of Tuberculosis Control. . . . It occurs to me that the most equitable way in which to obtain the money would be either from a gasoline tax or a tax on gas and oil and gas at its source, that is, a production tax. It is my understanding that approximately 80 per cent of the gas and oil is paid for by citizens outside of the State of Texas, yet our National resources are being depleted, by having these resources leave the State of Texas. A tax such as this would give a definite sum per year upon which to rely and you would not be subject to the whim of the Legislature each year. It is my opinion, however, when you go before the Legislature with a Tuberculosis Control Program, you should have some concrete suggestion, as to where the money might come from; otherwise, you would have just as strong a fight against the sources of the appropriation as you would the appropriation itself regardless of the source."

To educate the voters and public officials of the State to the need of adequate tuberculosis control, leadership will be required, comparable to Community Chest Drives or Government Bond Sales, to organize workers and clubs in every county of the State.

It would also be wise to employ a Commission, composed of an architect with experience in construction of tuberculosis hospitals, an officer of the U. S. Public Health Service, a tuberculosis specialist with experience in the administration of State District hospitals, and a tuberculosis specialist with experience in tuberculosis control where county hospitals are used. Such expert advice is of material value in planning. Their recommendations would lend support for legislative requests for specific appropriations.

Demonstration of Tuberculosis Control

A demonstration that was made as an experiment in 1917 by the Metropolitan Life Insurance Company was as follows:

This company offered to the National Tuberculosis Association the sum of $100,000 for the purpose of demonstrating, if possible, the control of tuberculosis in a typical American community by applying the knowledge of medicine and methods of tuberculosis

control. After some research, Framingham, Massachusetts, a town of approximately 17,000 population was selected for this purpose. The original appropriation was practically doubled in the course of the demonstration which covered a period of seven years—beginning in 1917.

The reports of this demonstration are set forth in a series of ten monographs, the last of which—number ten—gives the final summary report. This demonstration was under the direction of Dr. Donald B. Armstrong.

A preliminary survey of a sickness census, taken through a home canvass, disclosed a rate of 1.8 per cent for incapacitating sickness, 6.2 per cent for total illness, and .24 per cent for tuberculosis in a home canvass of 6,582 people of the town.

The establishment of a diagnostic and consultation service for physicians had two marked effects in case findings; namely, an actual increase in the number of cases, and a marked increase in the percentage of incipient tuberculosis discovered. Eventually, the ratio of active cases to annual deaths from tuberculosis was found to be nine to one. In other words, wherever there is one death caused from tuberculosis, one may expect to find approximately nine active cases of tuberculosis. It was also found that one per cent of the population of this typical town had active tuberculosis and, also, one per cent of the population were arrested cases of tuberculosis. It was also found that the percentage of the cases discovered in the early stages increased from fifty-five per cent to eighty-eight per cent, while the cases reported before death increased from 60 to 93 per cent. The percentage of cases treated in institutions increased from 15 to over 50 per cent. The experience of the demonstration indicated that from one to two institutional beds were necessary for every annual death of tuberculosis.

The most significant result of this attempt at tuberculosis control was the way in which community sentiment was aroused so that this small town was willing to pay for tuberculosis control. The cost of health work in the community for all private agencies increased during the seven years from $900 to $17,000 per year. The total cost of health work in the community for all agencies increased from $0.40 per capita per year to $2.40 per capita per year. These figures indicate that lack of funds can be overcome, provided proper leadership and education is available.

As an example of what can be done in the control and ultimate eradication of human tuberculosis, we may well profit by the example set and experience obtained by the Live Stock Industry in obtaining legislation.

H. R. Smith, General Manager, National Live Stock Loss Prevention Board, stated in an article in 1944:

"On March 4, 1917, the Congress appropriated $75,000 for tuberculosis eradication, but no provision was made for indemnity on reacting cattle slaughtered."

"In 1917 we supplied information to Congressman C. H. Sloan of Nebraska, author of a bill providing an appropriation of $1,000,000 for tuberculosis eradication. At the first hearing before the House Agricultural Committee, January 14, 1918, the writer stated that progress could not be made without partial reimbursement on reacting cattle slaughtered, so large a number of cattle were then infected, many breeders would otherwise be put out of business. It was pointed out that this was also a public health measure and every taxpayer should be willing to help. A clause was inserted to provide that the Federal Government would pay one-third of the loss to a limit of $25.00 on grades and $50.00 on pure-breds, provided the state or county would pay at least an equal amount. Soon after, numerous state legislative hearings were held and appropriations made out to meet the conditions of the Federal Laws."

"The House of Representatives voted $250,000, the amount recommended by the U. S. Department of Agriculture. Resolutions adopted by a number of livestock organizations, urging a larger appropriation, were sent and the Senate increased this to $500,000 for indemnity and operating expense, which, after a public hearing attended by livestock men from the various states, was later approved by the House."

"Each year, thereafter, hearings were held in Washington attended by representatives of producer organizations, market groups, State Departments of Agriculture and medical associations. Charts were prepared to show the progress in cattle testing under the able direction of the U. S. Bureau of Animal Industry and the state departments, with the resultant decrease in losses. These annual hearings were continued until 1928 when Congress appropriated $6,000,000. By 1932 this was increased to $6,505,800. The state and county appropriations totaled approximately $13,000,000 for that year. By 1935, the Federal, State, and County appropriations totaled $26,792,179—the maximum amount for any year with substantial decreases since, according to needs, until now a relatively small sum is required."

SUMMARY

1) The essentials and a pattern of tuberculosis control has been discussed.

2) Legislation for the correction of division of authority and conflicts that may arise is advised.

3) The present-day cost of construction and maintenance of 4,000 additional beds has been estimated.

4) The question as to wether the State and counties shall share, and share alike, is open for future decision, but that the State share half of the per-diem cost per patient is definitely advised where acceptable county and city tuberculosis hospitals exist, and until such time as District State Hospitals are built.

5) In carrying out one of the essentials of adequate tuberculosis control, to wit: X-raying of the entire population; employment of personnel, such as part-time or full time tuberculosis specialists and field nurses; and the establishment of tuberculosis pneumothorax and diagnostic clinics, well over $500,000 should be appropriated annually to the State Department of Health, earmarked for the Division of Tuberculosis—this is necessary.

6) Legislation for large appropriations of money is indicated and can be justified from an economic standpoint by the data and figures submitted.

7) An educational program for the purpose of better tuberculosis control is advocated and the appointment of a Commission composed of an architect and three medical experts in tuberculosis control to study and report their findings, is recommended.

RESUMEN

1) Se han discutido los rasgos esenciales y el patrón para el control de la tuberculosis.

2) Se recomienda que se expidan leyes para la corrección de la división de autoridad y de los conflictos que puedan ocurrir.

3) Se ha calculado el costo anual de la construcción y el mantenimiento de 4,000 camas adicionales.

4) La cuestión de si el Estado y los Condados deban contribuir, y contribuir igualmente, ha de ser decidida en el futuro; pero se recomienda definitivamente que el Estado contribuya con la mitad del costo diario por paciente donde existan hospitales aceptables en el condado y en el municipio y hasta tanto se construyan Hospitales Distritoriales del Estado.

5) Para llevar a cabo uno de los puntos esenciales del control adecuado de la tuberculosis, a saber, exámenes radiográficos de la población entera; empleo de personal, tal como especialistas de tuberculosis y enfermeras de campo, empleados parte del tiempo o de tiempo completo; y el establecimiento de clínicas para el diagnóstico de la tuberculosis y la administración de neumotórax, debe asignarse anualmente al Departamento de Sanidad del Es-

tado una partida de mucho más de $500,000, destinada para la División de Tuberculosis. Esto es necesario.

6) Es necesario que se expidan leyes que adjudiquen grandes sumas de dinero, lo que se justifica desde el punto de vista económico por los datos y cifras presentados.

7) Se aboga por un programa educativo con el fin de lograr un mejor control de la tuberculosis, y se recomienda el nombramiento de una Comisión compuesta de un arquitecto y de tres médicos expertos en el control de la tuberculosis para que conduzcan un estudio y presenten sus hallazgos.

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Necropsy Incidence of Tuberculosis in a Hospital for the Mentally Ill*

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The high incidence of tuberculosis in mental institutions has received wide attention in the literature of recent years. A number of authors have pointed out that deaths due to tuberculosis are far more numerous in patients in mental institutions than they are in the general population. Kupka[1] stated that about 5 per cent of all tuberculosis deaths occur in mental institutions. Deegan and his co-workers[2] reported that for a three year period (1937-1939) the death rate for tuberculosis in mental hospitals in the state of New York was 12 times the tuberculosis death rate of the general population of the state.

Most of these estimates are based on clinical diagnoses, and statistical reports on the incidence of tuberculosis in mental hospitals based on autopsy records have not been adequate or conclusive. It is felt that such data are necessary for a complete understanding of the problem of tuberculosis in mental hospitals, so that proper measures may be adopted for the prevention of spread of the disease to both patients and employees.

The present report is based on a series of 634 consecutive autopsies performed at the Pittsburgh City Home and Hospitals, Mayview, Pennsylvania, over a five year period from 1937 to 1941 inclusive. This series is unusual in that it includes both general hospital and mental patients, and it affords an opportunity for some comparison of the two groups. The general hospital statistics at this institution cannot be compared to the average general hospital records, however, as most of the general hospital patients admitted were suffering from long standing, chronic, medical conditions. Only a few surgical patients are included in the group.

The institution had a small tuberculosis hospital in a separate building with facilities for both general and mental hospital patients, so that patients with a clinical diagnosis of tuberculosis were transferred to this department and the records of the deaths in this group are included in our statistics.

For the purpose of this report, healed tuberculosis was not included in our study. We included active tuberculosis anywhere in the body, although we were primarily interested in active tuber-

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culosis of the lungs from the standpoint of determining the amount of exposure to which other persons might have been subjected. The term tuberculosis in this report is used only to indicate active tuberculosis.

The incidence of tuberculosis was high in both mental and non-mental groups. Active tuberculosis was found in 52 of 245 necropsies on mental patients, an incidence of 21.2 per cent, and in 59 of 389 necropsies on general hospital patients, an incidence of 15.1 per cent. The high incidence of tuberculosis in general hospital patients is at least partially explained by the high percentage of chronic medical cases admitted to the hospital and by the fact that when tuberculosis was diagnosed the patients were kept in the institution. Even so, the frequency of tuberculosis in mental patients was 40.3 per cent higher than in general hospital patients.

This study confirms the frequently reported observation that tuberculosis is not exclusively a disease of young people. The average age of mental patients with tuberculosis was 51.9 years, while the average age of the general hospital patients with tuberculosis was 58.4 years. The average age at autopsy of the entire group was 60.04 years.

The incidence of tuberculosis according to sex did not appear significant. There were 472 males in the series with a tuberculosis incidence of 18.2 per cent and 162 females with a tuberculosis incidence of 15.4 per cent.

In view of the danger of spread of infection, particularly in the mental group, we were interested in the degree of accuracy of the clinical diagnoses in these cases. In the mental group 28 out of 52 (53.8 per cent) were diagnosed before death, and in the general hospital group 33 out of 59 (55.9 per cent) were diagnosed before death. In connection with the danger to other mental patients, we discovered that 11 of the mental patients with a correct diagnosis of tuberculosis before death were diagnosed only within a month of the date of their death. In other words, the condition was discovered only in the terminal stages, and the exposure to other patients was almost as great as if the clinical diagnosis had not been made. The percentage of correct diagnoses in the general hospital group might have been higher except that some of the general hospital patients were admitted to the hospital in a dying condition, so that in certain instances the time interval before death did not allow for proper evaluation of the case.

Tuberculosis was not the primary cause of death in all the 111 patients with active tuberculous lesions. We arbitrarily divided the group into three main divisions. In the first, tuberculosis was the primary cause of death, in the second, tuberculosis was regarded as contributory to the death, while in the third group,

tuberculosis did not contribute to the death. Among the mental patients 39 died of tuberculosis, a death rate for the group of 15.9 per cent. Of the general hospital patients 41 died of tuberculosis, a death rate of 10.5 per cent for this group. These figures are shown in Table 1.

TABLE 1
Classification of Tuberculosis in Relation to Cause of Death

Patients	Primary		Contributory		Non-Contributory	
	No.	Per cent	No.	Per cent	No.	Per cent
Mental	39	15.9	4	1.6	9	3.6
General	41	10.5	7	1.7	11	2.8
TOTAL	80		11		20	

The tuberculous patients were also divided into three groups in relation to the presence or activity of tuberculosis in the lungs. The first group included those patients with active tuberculosis in the lungs, the second included those with healed tuberculosis in the lungs and active tuberculosis elsewhere, and the third group was made up of those patients showing no tuberculosis in the lungs but active tuberculosis elsewhere. These figures are shown in Table 2. Of those listed as showing active tuberculosis of the

TABLE 2
Classification of Lesions

Patients	Active in Lungs		Obsolete in Lungs Active elsewhere	Not in Lungs Active elsewhere
	No.	Per cent		
Mental	52	21.2	0	0
General	53	13.6	3	3

lungs, five showed only minimal changes, while the others showed moderate to far advanced tuberculous involvement.

We have not attempted to present the figures on the incidence of active tuberculosis in locations outside the lungs. However, we considered the possibility that tuberculous enterocolitis might be more frequent in mental patients due either to voluntary swallowing of sputum or to involuntary swallowing of sputum due to interference with reflexes. In the group with tuberculosis of the lungs 37 or 35.2 per cent of the patients showed tuberculous enterocolitis, but there was no significant difference in incidence between mental and nonmental groups. Among the mental patients 19 or 36.5 per cent showed tuberculous enterocolitis, while 18 or 33.9 per cent of general hospital patients showed similar involvement.

SUMMARY

1) Active tuberculosis was found in a high percentage of mental and general hospital deaths in a series of 634 consecutive necropsies in a large city institution. Active tuberculosis was noted in 52 of 245 necropsies on mental patients and in 59 of 389 necropsies on general hospital patients.

2) The percentage of these patients with a clinical diagnosis of tuberculosis was not high. Among the mental group only 28 out of 52 were diagnosed before death and 11 of these 28 were diagnosed only within the month prior to death.

3) Statistics on the incidence of tuberculosis as the primary cause of death in these patients are presented. Tuberculosis was found to be the primary cause of death in 15.9 per cent of the mental patients and 10.5 per cent of the general hospital patients. These necropsy statistics substantiate current reports in the literature concerning the high death rate from tuberculosis in mental institutions. The study emphasizes the need for continued effort to attain proper diagnostic and preventive measures in such institutions.

RESUMEN

1) Se descubrió tuberculosis activa en un elevado porcentaje de defunciones entre los pacientes mentales y de hospital general en una serie de 634 autopsias consecutivas en una grande institución municipal. Se encontró tuberculosis activa en 52 de 245 autopsias en pacientes mentales fallecidos y en 59 de 389 autopsias en pacientes de hospital general.

2) No fue alto el porcentaje de estos pacientes en los que se había hecho un diagnóstico clínico de tuberculosis. Entre el grupo de enfermos mentales solamente 28 de los 52 fueron diagnosticados antes de morir y 11 de estos 28 sólo fueron diagnosticados durante el mes anterior a su muerte.

3) Se presentan datos estadísticos sobre la frecuencia de la tuberculosis como causa principal de muerte en estos pacientes. Se encontró que la tuberculosis fue la causa principal de muerte en el 15.9 por ciento de los enfermos mentales y del 10.5 por ciento de los pacientes de hospital general. Estas estadísticas autópsicas verifican los corrientes informes en la literatura acerca de la elevada mortalidad por tuberculosis en instituciones de enfermedades mentales. El estudio recalca la necesidad de continuar esforzándose por alcanzar apropiadas medidas diagnósticas y profilácticas en esas instituciones.

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EDITORIALS

Necropsy Diagnoses

In this issue of *Diseases of the Chest,* Moran reports that among 245 necropsies of persons who died in an institution for the mentally ill, 52 (21.2 per cent) had active tuberculosis, while among 389 who died in a general hospital 59 (15.1 per cent) had active disease. In the institution for the mentally ill only 28 (53.8 per cent) of the 52 and in the general hospital group, 33 (55.9 per cent) of the 59 cases were diagnosed before death. The average age of the entire 111 patients at the time of autopsy was 60.04 years. This is not surprising because the age at which clinical tuberculosis causes illness and death has shifted upward during the past three decades.

In an analysis of 14,719 necropsies in private and general hospitals in New York City and vicinity, Medlar compared those which were done from 1916 to 1920, with those between 1940 and 1945. Pathologically significant tuberculosis was less frequently found in those under 10 years of age in the 1940-1945 group. Thus, the autopsy findings would indicate that the measures used to protect infants and children in New York have been effective. This is also borne out in a striking way in mortality rates. For example, in 1915 among children under 15 years, 1200 died from tuberculosis, whereas, in 1940 only 110. The population in this age period has remained fairly constant at 1,500,000. Moreover, the infection attack rate among children has markedly decreased. Obviously, where there is no primary tuberculosis there can be no endogenous reinfections resulting in illness and death. Children have been so well protected that a relatively small number now have primary tuberculosis, and a correspondingly low incidence of pathologically significant lesions are seen at necropsy. This situation can be observed wherever children have been protected against tuberculosis. The longer these protective measures have been in existence, the farther into life's span this low incidence of tuberculosis extends. In the Midwest and West of the United States there are areas where less than 10 per cent of those of 19 years react to tuberculin. Thus, more than 90 per cent have escaped primary lesions and, obviously, the incidence of pathologically significant tuberculosis found at necropsy in this age period is extremely low.

Between 10 and 50 years there was no essential difference, but among persons who died after the age of 50 years, Medlar found more pathologically significant tuberculosis in those examined from 1940 to 1945 than in those from 1916 to 1920. This is not surprising for two reasons: (1) There is probably as much residual tuberculosis among humans in this age group now as among those of the same age from 1916 to 1920. Probably both groups had approximately the same exposure in childhood and young adulthood. Certainly those now over 50 years were afforded little protection against the human and the bovine types of tubercle bacilli as infants or children. Therefore, the majority of them probably developed primary lesions, and in some there still reside the descendants of the tubercle bacilli which initially infected them. (2) The lives of many more persons extend into the period beyond 50 years than was true from 1916 to 1920, and thus more tuberculous lesions

have time to mature. Therefore, one would expect to find more clinically and pathologically significant tuberculosis among the old people of today than among those of a quarter of a century ago. Moran and Medlar emphasize the great importance of adequate examination of all persons beyond the age of 50 years as the proper disposition of those with contagious disease is an essential part of the tuberculosis control program of any community.

Much could be contributed to our knowledge of tuberculosis by more thoroughgoing pathological studies such as those by Moran and Medlar. The pathologist is the last court of appeal in diagnosis, since he has opportunities afforded no one else in the field. There is nothing known from naked eye inspection, through bacteriological determinations, microscopic studies and the most delicate chemical tests that he cannot use in a direct manner. The superiority of his diagnostic armamentarium places upon the pathologist a responsibility shared by no other person in the field of diagnosis. Unless he uses every facility at his command his diagnoses can be just as erroneous as those made antemortem by physicians and surgeons. Errors by pathologists may be more serious than those of others from the standpoint of vital statistics, contagion, litigation, etc., because their diagnoses are regarded as final.

The pathologist also has the unique opportunity of locating previously unsuspected conditions and diagnosing them accurately. This is particularly true of a vicissitudinous disease like tuberculosis whose lesions may alternately be active, quiescent or arrested, temporarily or permanently. They may be so small as to cause no pressure symptoms or toxemia. Again, if in the lungs they may attain extensive proportions without causing pain, since these organs are devoid of sensory pain nerve fibers. They may cause no shortness of breath because of the large reserve capacity of the lungs. Therefore, these lesions often are not diagnosed or even suspected until the pathologist finds them. Wherever tuberculous infection is universal one expects the pathologist to find tuberculous lesions in the bodies of all adults dead from any cause. These lesions vary, from the size of a pinhead to massive excavated processes.

The physician has only two methods of procuring accurate information regarding the incidence of tuberculosis in any group of people, namely, the tuberculin reaction and the postmortem examination. The tuberculin reaction, properly administered, should coincide with postmortem findings. This is as true in man as in domestic or experimental animals. No phase of the examination except the tuberculin test is delicate enough to reveal anything more than gross pathology in the living body. If careful antemortem examinations are made, including x-ray film inspection of the chest, but the tuberculin test is omitted, there will be many surprises at the postmortem table. In fact, a great deal of tuberculosis can exist in tuberculin reactors which is not located by any phase of the examination during life but is clearly in evidence at postmortem.

The student of tuberculosis today recognizes the absurdity of attempts to make accurate diagnoses of tuberculosis from x-ray shadows alone. He is also aware of the fact that pathologists' diagnoses without adequate investigation can be just as erroneous as those made from x-ray shadows. A famous pathologist was asked what percentage of the calcium deposits found at necropsy in the pulmonary parenchyma and

hilum regions are due to tuberculosis. His answer was 99.5 per cent. Obviously he had never taken the time to determine the etiological agents producing lesions in which calcium is deposited. If he had, in all probability he would have discovered that more than half of the depositions of calcium which he assumed resulted from tuberculosis were actually due to other conditions, with a preponderance of fungus infections, because he worked in an area where histoplasmosis was later found to be endemic.

Long ago pathologists had the opportunity to discover that coccidioidomycosis frequently has calcium deposit in its lesions, but they did not take time to make sufficiently detailed examinations and thus went on believing and teaching that tuberculosis is responsible for most of the calcium deposits in the lungs. Inaccuracies in pathological diagnoses also extended to other types of pulmonary lesions, even those that had cavitated and were extensive.

Today the student of tuberculosis does not give serious consideration to a pathological diagnosis unless bacteriological evidence has been obtained, not just by finding acid-fast bacilli and giant cells in smears and sections, but by culture and, still better, by animal inoculation. He does not accept the diagnosis of any other disease caused by microorganisms unless they are actually recovered from materials obtained at postmortem examination. He does not accept the diagnosis of silicosis unless chemical analysis reveals the presence of free silica in sufficient quantity to produce this disease. In this era, when specific evidence is demanded before the pathologist's diagnosis is acceptable, one wonders about the accuracy of many diagnoses in the past, resulting in such broad and sweeping statements concerning so called epidemics of acute tuberculosis.

J. A. M.

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Carlo Forlanini — 1847-1918

In commemoration of the 100th anniversary of his birth

One hundred years have gone by since the birth of Carlo Forlanini, the originator of the systematic use of artificial pneumothorax as an independent therapeutic measure for the treatment of pulmonary tuberculosis. Born in Italy, he completed his medical studies at the age of 23. Then he joined the staff of the Ospedale Maggiore of Milan. His clinical ability was given due recognition by his promotion to the position of chief of the medical, pediatric, obstetrical and dermatologic services at this hospital. In 1884, at the age of 37, he began his teaching activities in physical diagnosis at the University of Turin and continued at the University of Pavia in 1899. At the latter institution, he became Professor of Clinical Medicine in 1900. During his entire professional career, he devoted a great deal of time and effort to the study of tuberculosis and contributed extensively to the medical literature on this subject. The year of 1882 is a memorable one in the history of Medicine on account of the discovery of the tubercle bacillus by Koch and because of the publication of Forlanini's epoch-making treatise on the rationale of

artificial pneumothorax. The latter was printed in the Gazetta degli Ospedali & delle Cliniche di Milano, Volume 3, No. 68, Page 537, August 23 and in subsequent issues of the same magazine during the same year. His reasoning was based on the observations of other clinicians as well as of his own, noticing that spontaneous pneumothorax, with or without pleural effusion, had a favorable effect on the course of pulmonary tuberculosis. He remarked: "Surgeons have either let air enter the pleura, or have injected it for therapeutic purposes, both with no harmful consequences; if a pneumothorax arrests, as such, the course of phthisis in a lung, if hydrothorax or pyothorax have formed by way of pulmonary paths; why should we not cause in a consumptive an artificial pneumothorax through the chest wall, with the necessary precautions (easy to obtain) that will be useful in preventing secondary pleuritic processes?"

As to the technique of artificial pneumothorax, he pointed out that "it is necessary that the volume of air and its pressure be such as not to displace the mediastinum, and,—whether the pulmonary opening stay open or be closed,—that inspiration would not bring the pressure down to a level insufficient to balance the pulmonary elasticity, 5-7 mm. of mercury in normal conditions, and certainly a little more when the lung is consumptive. If the pressure of the pleural air goes above this level, the patient becomes dyspneic, suffers from it, and may eventually die rapidly by suffocation; if, instead it goes below the level, the lung participates in the respiratory motions of the thoracic box, and so the mechanical reason for the beneficial action of pneumothorax is missed or lost."

The basic idea of his concept was that artificial pneumothorax was useful in tuberculosis because it immobilized the lung. It is true that the thought of compressing the lung during intrapleural pneumothorax treatment has been entirely abandoned. But artificial pneumothorax still enjoys an unprecedented preference by physicians engaged in the treatment of pulmonary tuberculosis. Appropriately, today the so-called low-tension or hypotensive pneumothorax is the method of choice. This procedure entails all the possible advantages without the risks of other types of pneumothorax. The healing which results from the use of artificial pneumothorax is predicated upon relaxation of the lung. This organ is richly supplied with elastic fibers. This implies an innate pulmonary elasticity which causes the lung to recoil toward the hilum during each expiration and also, when the lung is free of the influence of the normal intrapleural negative pressure. In this sense, the pneumothorax lung is a relaxed organ as compared with its stretched-out condition under physiologic circumstances. Maybe this is an opportune moment to advance a plea for changing the terminology pertaining to artificial pneumothorax and other conservative methods which aim at a similar mechanical change in the diseased lung. It is proposed to discard the term collapse therapy. In its connotation, collapse carries the meaning of abrupt shrinking or falling together, with utter failure and uselessness. It is agreed that such changes in the treated lung do not come under the intended purpose of artificial pneumothorax. As a matter of fact, an adequate pneumothorax calls for a gradual and moderate reduction in the size of the lung. In the true sense of the word, this is a state of relaxation and not collapse. Thus, it appears that relaxation therapy is a more expressive term for portraying the effect of an ideal pneumothorax on the lung.

The working mechanics of artificial pneumothorax readily explain its potential therapeutic benefits. The intrapleural negative pressure is the expression of the difference between the retractile elasticity of the lung and the traction exerted upon it by the thoracic cage (including the diaphragm) through the cohesion of the visceral and parietal pleurae. The thoracic cage is larger than the lung under physiologic circumstances. The interaction of the centripetal force of pulmonary contractility and the centrifugal force represented by the thoracic cage results in a force which exerts a suction effect upon the lung. It is interesting to recall in this connection that while the atmospheric pressure at sea level is 760 mm. of mercury, the average intrapleural pressure is only 5.1 mm. of mercury less than this on inspiration, and only 1.4 mm. less on expiration. This apparently insignificant difference represents the suction force which holds the lung in apposition to the inner surface of the thorax. As soon as the suction effect of the intrapleural negative pressure is rendered less negative by artificial pneumothorax, the force of elastic recoil of the lung comes into play.

The following factors are considered the most important in the healing of a tuberculous lesion under pneumothorax treatment: (1) mechanical closure of cavities; (2) lessening of bronchogenous, lymphogenous and hematogenous spread of the disease; (3) decrease in the absorption of toxic materials from the involved area; (4) fibrosis due to lymph stasis; (5) fibrosis due to relative tissue anoxia; (6) inhibition of the propagation of tubercle bacilli resulting from local tissue anoxia; (7) impairment of the viability of the tubercle bacilli in consequence of accumulation of carbon dioxide in some areas of the relaxed lung.

The selection of cases for artificial pneumothorax should be done with scrupulous discrimination. According to prevailing trends, most patients are given a reasonable trial with strict bed rest prior to the induction of pneumothorax. There are sound reasons for such attitude and only few exceptions to this program. Strict bed rest alone is a potent method in the treatment of patients with reversible forms of pulmonary tuberculosis. In addition to the obvious general benefits derived from this measure, it is well to bear in mind that a certain degree of reduction in the intrapleural suction can be accomplished by bed rest. It must be recognized that the stretch of the lung in the upright position (that I propose to designate as orthostatic hypertension of the elastic tissue of the lung) is ontogenetically unnatural. An effective reduction in the suction force of the intrapleural negative pressure is brought about by decreasing the difference between the size of the retractile lung and that of the thoracic cage. Normally, the lung automatically adjusts itself to the size of the thorax. In the recumbent position the size of the thoracic cage is reduced. Consequently, there is a decrease in the size of the lung. It is known that in the supine position the lung volume is reduced by 20 per cent. Herein lies the mechanical basis of the rationale of bed rest in the treatment of pulmonary tuberculosis.

An ideal pneumothorax must be mechanically satisfactory. Adhesions which prevent competent pulmonary relaxation should be severed as early as possible during the course of treatment. It is the consensus that a mechanically unsatisfactory pneumothorax is dangerous on account of its possible complications. It should never be forgotten that a mechanically unsatisfactory pneumothorax is *eo ipso* therapeutically

ineffective and therefore, it should be looked upon as a hazardous venture, with the patient's life at stake.

Remarkable as its curative results are, it may be necessary to supplement artificial pneumothorax with other forms of relaxation therapy. Good clinical judgment, resourcefulness and freedom from therapeutic prejudice are prerequisites in this regard. Only by being cognizant of the benefits as well as the limitations of artificial pneumothorax can a good clinician use this measure to the best advantage of the tuberculous patient.

A purview of the modern management of pulmonary tuberculosis permits us to say that the universal acceptance of artificial pneumothorax in the treatment of this condition is the greatest monument the medical profession could ever have erected to the genius of Carlo Forlanini.

A. L. B.

REFERENCE

Forlanini, C.: "A Contribution to the Surgical Therapy of Phthisis. Ablation of the Lung? Artificial Pneumothorax?. *Gazetta degli Ospedali & delle Cliniche di Milano*, 3:537, 585, 601, 609, 617, 625, 641, 657, 665, 689, 705, 1882; translated by Lojacono, S., *Tubercle* 16:61, 1934.

A New Medical Journal

The first issue of a new medical journal has just appeared under the title, REVISTA PANAMERICANA DE MEDICINA Y CIRUGIA DEL TORAX. The Editor, Donato G. Alarcon, M.D., F.C.C.P., of Mexico City, is widely known on the Western Hemisphere and throughout the world for his excellent work in clinical medicine and surgery, as well as medical journalism. It is his desire to publish up-to-date material from South, Central and North America, thus keeping all physicians in the Western Hemisphere informed on the latest developments and procedures in the field of chest diseases. This is a fine and laudible venture in medical education. The value of the journal is enhanced for North American scientists and physicians by a summary in English at the end of each article. Indeed, every journal published on the Western Hemisphere definitely increases its usefulness by publishing summaries of all articles at least in Spanish and English.

The first copy of this excellent journal contains the following articles:

"El Tratamiento del Conducto Arterial Persistente" (The Treatment of Patent Ductus Arteriosus), by Frank S. Dolley, M.D., F.C.C.P., Los Angeles, California.

"La Iniciacion de la Tuberculosis en el Adulto" (The Onset of Tuberculosis in the Adult), by Gumersindo Sayago, M.D., F.C.C.P., Cordoba, Argentina.

"Concepto Sobre la Curacion de la Tuberculosis. Importancia del Lavado Bronquial como Indice de Ella. Tecnica" (The Importance of Bronchial Lavage in the Treatment of Tuberculosis; Describing a New Technique), by Juan J. Castillo, M.D., F.C.C.P., Havana, Cuba.

Some space is devoted to the Pan American activities of the American College of Chest Physicians. The illustrations and general format of the journal are of the highest quality.

It is fortunate that such an excellent physician and editor should be

so ideally located geographically to serve all inhabited areas from pole to pole.

Although there are several fine journals devoted to thoracic diseases published in various parts of this hemisphere there are not too many. Indeed, there is ample room for another, as indicated by the increasing demand for such space. DISEASES OF THE CHEST welcomes REVISTA PANAMERICANA DE MEDICINA Y CIRUGIA DEL TORAX and stands ready to cooperate with and support Dr. Alarcon and his editorial board wholeheartedly at every opportunity.

J. A. M.

14th Annual Meeting

AMERICAN COLLEGE OF CHEST PHYSICIANS

Congress Hotel, Chicago, Illinois

June 17-20, 1948

Chicago will be host this year to the American Medical Association and the American College of Chest Physicians, as well as to a number of other medical specialty societies that are planning to hold meetings in June. It is anticipated that there will be a large attendance and we therefore urge you to make your hotel reservations at once if you are planning to attend these meetings. Please write to the Executive Offices of the College in Chicago for hotel accommodations for the College meeting, June 17-20, and for the meeting of the American Medical Association, June 21-25. (See page xvi of this issue for hotel rates and reservation coupon).

The Program Committee assures us that the scientific program will maintain the high standards which are identified with College meetings. The program will be international in scope and topics of current interest will be discussed by leading authorities.

As previously announced, it has been voted by the House of Delegates of the American Medical Association that a Section be established on Diseases of the Chest. The first session for this newly organized Section will be presented at Chicago in June, 1948, under the Section on Miscellaneous Topics in the Scientific Assembly of the American Medical Association.

Richard H. Overholt, M.D., F.C.C.P., Brookline, Massachusetts, and J. Winthrop Peabody, M.D., F.C.C.P., Washington, D. C., have been appointed Chairman and Secretary, respectively, for this session.

All chest specialists are urged to register for the Section and attend these meetings.

RAHWAY, N. J.—Dr. Donald F. Robertson, Associate Medical Director of Merck & Co., Inc., manufacturing chemists of Rahway, N. J., died November 8th in Harkness Pavilion, Presbyterian Medical Center, New York City. A member of the Medical Division for more than ten years, Dr. Robertson joined the company after extensive postgraduate training in neurology and allied subjects. He contributed to the development of the Merck program of clinical research, and is credited particularly with important studies on penicillin and streptomycin.

Postgraduate Course in Diseases of the Chest

Sponsored by

THE AMERICAN COLLEGE OF CHEST PHYSICIANS

PENNSYLVANIA CHAPTER

and

THE LAENNEC SOCIETY OF PHILADELPHIA

March 15-20, 1948

Warwick Hotel, Philadelphia, Pennsylvania

A postgraduate course in diseases of the chest will be given by the American College of Chest Physicians, Pennsylvania Chapter, and the Laennec Society of Philadelphia, at the Warwick Hotel, Philadelphia, Pennsylvania, March 15-20, 1948.

This course is intended to present authoritative information on all of the important recent developments in the diagnosis and treatment of diseases of the chest. A feature of the course will be luncheon round table discussions, including question and answer periods on controversial matters. The subjects to be considered in the course are listed in the preliminary outline below.

To insure informality and individual attention, the class is limited to fifty members. The tuition fee is $50.00. When the class is filled, no further applications will be accepted. Requests for further information should be sent to the Executive Offices of the American College of Chest Physicians in Chicago. A coupon has been inserted on page x of the front pages of this issue to facilitate application.

Hotel accommodations at the Warwick Hotel in Philadelphia, will be arranged on request at the time of acceptance of applications.

— P R O G R A M —

Time: 9:00 a. m.:	
Monday:	Pulmonary Segments in Relation to Bronchopulmonary Diseases.
Tuesday:	Pneumoconiosis
Wednesday:	Anesthesiology in Relation to Thoracic Surgery with Special Reference to Pulmonary Complications.
Thursday:	Epidemiology and Control of Tuberculosis.
Friday:	Principles of Medical Treatment of Pulmonary Tuberculosis with Special Reference to Artificial Pneumothorax and Pneumoperitoneum.
Saturday:	Clinical Diagnostic Conference, Hahnemann Hospital; Staff.
Time: 10:00 a. m.:	
Monday:	Acute Respiratory Diseases.
Tuesday:	Bronchopulmonary Physiology.
Wednesday:	Allergic States in Bronchial and Pulmonary Conditions.
Thursday:	Diagnosis of Pulmonary Tuberculosis and Evaluation of Activity.
Friday:	Effects of Antibiotics in Tuberculosis.

Time: 11:00 a. m.:

Monday: Mycotic Infections of the Bronchi and Lungs.

Tuesday: Cardiac Factors in Chronic Pulmonary Disease.

Wednesday: Cardiac Surgery.

Thursday: Tuberculosis in Children.

Friday: Surgery of Pulmonary Tuberculosis.

Saturday: Clinical Diagnostic Conference, Jewish Hospital.

Time: 12 noon to 2:00 p. m. — Luncheon and Round Table Discussions:

Monday: The Diagnosis and Management of Pulmonary Abscess.

Tuesday: Diagnosis and Management of Pleural Effusions and Empyema.

Wednesday: B.C.G.; history; application; and discussion.

Thursday: Rehabilitation in Chronic Pulmonary conditions with Special Reference to Tuberculosis.

Friday: Mass X-ray Surveys.

Time: 2:00 p. m.:

Monday: Pathology of Chronic Bronchopulmonary Diseases.

Tuesday: Bronchiectasis

Wednesday: Bronchoscopy in Diagnosis and Treatment of Chronic Diseases of the Chest.

Thursday: Diagnostic and Therapeutic Clinic in Tuberculosis.

Friday: Psychosomatic Influences in Chronic Pulmonary Diseases.

Time: 3:00 p. m.:

Monday: Laboratory Methods in Diagnosis of Chest Diseases.

Tuesday: Surgical Treatment of Bronchogenic Carcinoma.

Wednesday: Pulmonary Embolism.

Thursday: The Use and Abuse of Rest in Pulmonary Tuberculosis.

Friday: Clinical Diagnostic Conference; and Demonstration of Tests for Cardio-Pulmonary Function (Barton Memorial Division, Jefferson Hospital).

Time: 4:00 p. m.:

Monday: Radiological Diagnosis of Acute and Chronic Pulmonary Diseases.

Tuesday: Mediastinal Tumors.

Wednesday: Aerosol Therapy in Chronic Bronchial Infections.

Thursday: Diagnosis and Surgical Treatment of Esophageal Disease.

Time: 5:00 p. m.: Clinical Diagnostic Conference, Temple University Hospital; Staff.

NOTE: The luncheon, round-table discussions and afternoon program on Thursday, March 18, will be presented at the Eagleville Sanatorium, Norristown, Pennsylvania.

Participants and instructors will be the guests of the sponsoring societies at a banquet on Friday night, March 19, at the Warwick Hotel. There will be a short program following the banquet and the subject will be "Literary Notes on Chest Diseases."

Board of Examiners

Candidates who passed the oral and written examinations for Fellowship conducted at Atlantic City, June 7, 1947:

Hans Abeles, M.D., New York, New York
Alfred Adler, M.D., Mount Vernon, Missouri
Joseph P. Atkins, M.D., Wynnewood, Pennsylvania
Ralph W. Ballin, M.D., State Sanatorium, Maryland
Solomon Bauch, M.D., Newark, New Jersey
Lewis F. Baum, M.D., South Orange, New Jersey
Otto S. Baum, M.D., South Orange, New Jersey
Johann S. Bornstein, M.D., Chicago, Illinois
Katherine Boucot, M.D., Philadelphia, Pennsylvania
J. Edmond Bryant, M.D., Evanston, Illinois
L. James Buis, M.D., Richmond, Virginia
Robert O. Canada, M.D., Washington, D. C.
Pasquale Ciaglia, M.D., Utica, New York
Joseph D. Cuono, M.D., Verona, New Jersey
Gisela K. Davidson, M.D., Portland, Maine
Richard Earle DeButts, M.D., Washington, D. C.
Thomas E. Dredge, M.D., Minneapolis, Minnesota
Benjamin Freedman, M.D., Wynantskill, New York
Edward M. Garment, M.D., Castle Point, New York
William H. Glass, M.D., Hartford, Connecticut
Leon Goldberg, M.D., New York, New York
J. Ivan Hershey, M.D., Bryn Mawr, Pennsylvania
John H. Hirschfeld, M.D., Baltimore, Maryland
Gerald F. Hogan, M.D., Amherst, Massachusetts
Isaac Horowitz, M.D., Brooklyn, New York
Hugh L. Houston, M.D., Murray, Kentucky
Leroy Hyde, M.D., Van Nuys, California
Charles Hyman, M.D., Atlantic City, New Jersey
Zohora Ismail, M.D., New York, New York
Benjamin Israel, M.D., New York, New York
Archibald R. Judd, M.D., Hamburg, Pennsylvania
Samuel Kahn, M.D., New York, New York
William S. Klein, M.D., Chicago, Illinois
Alexander Krasnitz, M.D., New Lisbon, New Jersey
Joseph D. Landesman, M.D., Corpus Christi, Texas
Anthony D. Lioy, M.D., Oteen, North Carolina
Harold A. Lyons, M.D., Washington, D. C.
David Marcus, M.D., Cleveland, Ohio
James W. Macdonald, M.D., Pittsburgh, Pennsylvania
Patrick I. McShane, M.D., Washington, D. C.
Constantine P. Mehas, M.D., Pontiac, Michigan
Solomon Netzer, M.D., San Francisco, California
William K. Nutzman, M.D., Kearney, Nebraska
John A. O'Hale, M.D., Brecksville, Ohio
Francis H. O'Neill, M.D., Pittsburgh, Pennsylvania
Rowland S. Philips, M.D., Glenn Dale, Maryland
Samuel Phillips, M.D., Memphis, Tennessee
Sam Poller, M.D., Castle Point, New York
Solomon M. Rauchwerger, M.D., Oteen, North Carolina
Robert E. Schell, M.D., Swannanoa, North Carolina
Joseph Schmerler, M.D., Liberty, New York
Reuben I. Shapiro, M.D., Detroit, Michigan
Irving S. Shiner, M.D., New York, New York
Harry Shubin, M.D., Philadelphia, Pennsylvania
Robert M. Sonneborn, M.D., Wheeling, West Virginia
J. Earle Stuart, M.D., Plainfield, New Jersey
John T. Szypulski, M.D., Mt. Carmel, Pennsylvania
Theodore J. Talbot, M.D., Staten Island, New York
Joseph F. Tedesco, M.D., Castle Point, New York
Samuel Topperman, M.D., Otisville, New York
Albert B. Tucker, M.D., Newark, New Jersey
Kenneth A. Tyler, M.D., Gooding, Idaho

Joseph L. Versage, M.D., Stockertown, Pennsylvania
Arthur John Viehman, M.D., Lexington, Kentucky
Arthur D. Ward, M.D., Worcester, Massachusetts
Charles M. Webster, M.D., The Dalles, Oregon
Cuthbert B. Young, M.D., Tyler, Texas

The questions used in the written examinations were as follows:

THORACIC DISEASES (MEDICAL ASPECTS)

Part I

Discuss the pathogenesis of atelectasis.

THORACIC DISEASES (MEDICAL ASPECTS)

(Answer any 4 of the 5 questions)

Part II

(1) Discuss the differential diagnosis between bronchiectasis and carcinoma of the bronchus.
(2) Outline the present day accepted treatment of lung abscess.
(3) Discuss tracheobronchial tuberculosis from the standpoint of pathogenesis, diagnosis and treatment.
(4) Discuss the etiological aspects of pleural effusion.
(5) Discuss evaluation of the effectiveness of artificial pneumothorax and the optimal time for its discontinuance.

THORACIC DISEASES (SURGICAL ASPECTS)

(Answer any 3 of the following 5 questions)

(1) In a suspected case of bronchiogenic carcinoma, what diagnostic methods would be used to confirm or exclude the diagnosis?
(2) What are the indications for pulmonary resection in tuberculosis?
(3) What surgical procedures are of value in bronchiectasis?
(4) Discuss the advantages and disadvantages of phrenicectomy.
(5) Discuss the differential diagnosis of a shadow in the upper anterior mediastinum. How would you arrive at a diagnosis?

BACTERIOLOGY AND IMMUNOLOGY

(Answer 3 of the following 5 questions)

(1) Name five species of acid fast bacilli other than mycobacterium tuberculosis and discuss their pathogenicity.
(2) How could you differentiate virulent human type tubercle bacilli from other acid fast bacilli?
(3) Discuss the physical and chemical mechanisms involved in acid fast staining.
(4) Outline the chemical composition of the tubercle bacillus and the properties of its chief constituents.
(5) Describe the growth of tubercle bacilli on artificial media.

PATHOLOGY

(Answer any 3 of the following 5 questions)

(1) Discuss the pathogenesis of lung abscess developing as a complication of acute appendicitis.
(2) Describe the gross and microscopic lesions in the lung in a case of chronic bronchiectasis.
(3) If a child aspirates a foreign body into the primary bronchus, what succession of pathological processes, structural and functional, would likely develop?

(4) Discuss the mechanism of the process of resolution of lobar pneumonia.
(5) What are the chief differences between the usual findings in pulmonary tuberculosis in a child and in an adult?

PHYSIOLOGY

(Answer any 3 of the following 4 questions)

(1) Define and give normal values for Tidal air, Complemental air, Supplemental air, Vital capacity, Residual air.
(2) Discuss leucocytic changes in the course of tuberculosis.
(3) Describe two techniques for determining sedimentation rate, and the normal values for each, and list conditions accelerating and retarding sedimentation of red corpuscles.
(4) Outline the chemical and the reflex mechanisms controlling respiration.

ANATOMY OF THE CHEST

(Answer any 3 of the following 5 questions)

(1) Give the surface markings on the exterior chest wall which outline the location of the underlying lungs and the individual lobes.
(2) Outline briefly the lymphatic drainage of the lungs.
(3) Describe the pleural layers and give their nerve supply.
(4) Give the boundaries and contents of the various parts of the mediastinum.
(5) Discuss the anatomy and the relations of the phrenic nerve.

Semi-Annual Meeting, Board of Regents

The Board of Regents of the American College of Chest Physicians met in Washington, D. C., at the Statler Hotel, November 21 and 22, 1947. Oral and written examinations for Fellowship in the College were given on Friday, November 21; various councils and committees held meetings during the two-day session, and the Board of Regents held their semi-annual meeting on Saturday, November 22.

Regents and other officials of the College present for the sessions were:

J. C. Placak, M.D., Cleveland, Ohio, Chairman
Andrew L. Banyai, M.D., Milwaukee, Wisconsin
Dean B. Cole, M.D., Richmond, Virginia
A. Barklie Coulter, M.D., Washington, D. C.
M. Jay Flipse, M.D., Miami, Florida
Carl H. Gellenthien, M.D., Valmora, New Mexico
Edward A. Greco, M.D., Portland, Maine
Alvis E. Greer, M.D., Houston, Texas
E. C. Harper, M.D., Richmond, Virginia
Edward W. Hayes, M.D., Monrovia, California
Charles M. Hendricks, M.D., El Paso, Texas
J. R. Herradora, M.D., Jersey City, New Jersey
William A. Hudson, M.D., Detroit, Michigan
Minas Joannides, M.D., Chicago, Illinois
Hollis E. Johnson, M.D., Nashville, Tennessee
Herbert L. Mantz, M.D., Kansas City, Missouri
C. Howard Marcy, M.D., Pittsburgh, Pennsylvania
Louis Mark, M.D., Columbus, Ohio
S. U. Marietta, M.D., Washington, D. C.
Edgar Mayer, M.D., New York, New York

Jay Arthur Myers, M.D., Minneapolis, Minnesota
George G. Ornstein, M.D., New York, New York
Richard H. Overholt, M.D., Brookline, Massachusetts
J. Winthrop Peabody, M.D., Washington, D. C.
Karl Schaffle, M.D., Asheville, North Carolina
Nelson W. Strohm, M.D., Buffalo, New York
James H. Stygall, M.D., Indianapolis, Indiana
Paul A. Turner, M.D., Louisville, Kentucky
Walter E. Vest, M.D., Huntington, West Virginia
Harry C. Warren, M.D., San Francisco, California
Francis J. Weber, M.D., Washington, D. C.
Roy A. Wolford, M.D., Washington, D. C.

Murray Kornfeld, Chicago, Illinois, Executive Secretary
Harriet E. Lumm, Chicago, Illinois, Asst. Executive Secy.

The reports and discussions presented at the meeting of the Board of Regents are given in the following agenda:

Report of the President,
Major General S. U. Marietta, Washington, D. C.

General Marietta in his opening remarks, discussed in detail the newly created Section on Diseases of the Chest in the Scientific Assembly of the American Medical Association and the steps that were being taken by the College Committee on Certification in their efforts to establish an acceptable Board on Diseases of the Chest.

Council on Undergraduate Medical Education,
Edward W. Hayes, M.D., Monrovia, California, Chairman.

The Council on Undergraduate Medical Education reported that the copy for the book entitled "The Fundamentals of Pulmonary Tuberculosis and Its Complications, for the Student, the Teacher, and the Practicing Physician," has been completed and it is expected that the book will be released by Charles C. Thomas Publishers in April, 1948. The copy for the book on non-tuberculous diseases of the chest is now in preparation. *Report adopted.*

Council on Postgraduate Medical Education,
J. Winthrop Peabody, M.D., Washington, D. C., Chairman.

The Council on Postgraduate Medical Education reported on the postgraduate course in diseases of the chest held at the Municipal Tuberculosis Sanitarium, Chicago, Illinois, September 15-20, 1947. This course was attended by 62 physicians representing 20 states, the District of Columbia, Canada, China, Cuba, Mexico and Spain. A similar course in diseases of the chest will be given at Philadelphia, March 15-20, 1948. *Report adopted.*

Editorial Board,
Jay Arthur Myers, M.D., Minneapolis, Minnesota, Chairman.

The Editorial Board reported that the number of pages in the College journal "Diseases of the Chest" have been increased, but despite this, there are a large number of manuscripts still to be published. It was recommended that authors be requested to condense the text and limit the number of illustrations of their papers. It was pointed out that the cost of publishing the journal has risen to such a great extent, it will be impossible in the future for the College to pay for more than four illustrations for any one paper. *Report adopted.*

Membership Committee,
Roy A. Wolford, M.D., Washington, D. C., Chairman.

The Membership Committee reported publication of the 1948 College Directory containing the listing of 2274 members. This is the seventh issue of the Directory and the first to be published since 1943. At that time the Directory contained the names of 1255 members of the College. Since the 1948 Directory was published, 153 physicians have been admitted to membership, bringing the present College membership to 2427, an increase of almost 100 per cent since 1943. The need of obtaining more Associate Fellows was stressed. Members are to be requested to give particular attention to this type of applicant. *Report adopted.*

Report of the Treasurer,
Benjamin L. Brock, M.D., Louisville, Kentucky.

The semi-annual financial report was referred to the Executive Council of the College. The annual report of the Treasurer has been published in "Diseases of the Chest" (Vol. XIII, No. 5, September-October, 1947, pages 534-536).

Resolution:
Since the College Board of Regents now meets twice each year, one of these meetings generally being held in November, your Treasurer wishes to recommend that the fiscal year be changed so that the books may be closed on December 31, rather than on April 30. I would further propose that the arrangements be made with the College Auditor to have this change made at the earliest possible date. *Report and Resolution adopted.*

National Council of Tuberculosis Committees,
James H. Stygall, M.D., Indianapolis, Indiana, Chairman.

The National Council of Tuberculosis Committees reported the enthusiastic luncheon meeting which was held in Atlantic City, June 8, at the time of the annual meeting of the College. It is planned to hold a similar meeting in Chicago in June, 1948. *Report adopted.*

Council on Public Health,
Paul A. Turner, M.D., Louisville, Kentucky.

Sub-Committee on Occupational Diseases of the Chest,
Louis Mark, M.D., Columbus, Ohio, Chairman.

The Committee on Occupational Diseases of the Chest, which serves under the Council on Public Health, presented a progress report. *Report adopted.*

Sub-Committee on Chest Diseases in Penal and Menial Institutions,
Otto L. Bettag, M.D., Pontiac, Illinois, Chairman.

The Committee on Chest Diseases in Penal and Mental Institutions, which serves under the Council on Public Health, reported on a meeting of the Committee held in Columbus, Ohio, on November 17, 1947.

The following Committee members were present at the meeting: Otto L. Bettag, M.D., Pontiac, Illinois, Chairman; B. B. Bagby, M.D., Swannanoa, North Carolina; George Curtis, M.D., Columbus, Ohio; and J. George Lang, M.D., New York, N. Y. Non-committee members present at the meeting were: Oren A. Beatty, M.D., Mansfield, Ohio; John V. Horst, M.D., Mansfield, Ohio; Louis Mark, M.D., Columbus, Ohio; M. S. Lauder,

M.D., Charleston, West Virginia; and Irville Spencer Rian, M.D., Mount Vernon, Ohio.

A study was made of the past, present and future work, as well as a review of the progress of the Committee. After open discussion the following recommendations were made:

1. Contact the Veterans Administration for a tuberculosis survey among the mental patients.
2. Contact the Federal Penal System for a similar view.
3. Continue to elicit the support of the American Prison Association, American Psychiatric Association and other national agencies interested in these special groups and public health.
4. Ask the Board of Regents to appoint to the Committee a member of the U. S. Public Health Service with special interest, training and experience in the immediate problem.
5. Bring the literature on this special subject up to date.

The success of the meeting suggests further sectional conferences to invite discussions of the problems in that locale. The next meeting of the Committee will be held in Chicago in June, 1948.

Invitations have been received from the Illinois Department of Public Safety and the Illinois Department of Public Welfare to visit the chest centers of these departments during the annual meeting of the College. All other interested physicians have also been invited. *Report adopted.*

Board of Examiners,
Alvis E. Greer, M.D., Houston, Texas, Chairman.

The Board of Examiners reported that 67 of the 72 candidates who took their examinations for Fellowship in the College in Atlantic City on June 5, have passed successfully. The names of the successful candidates are published in this issue of the journal. Nine physicians took their examinations for Fellowship in Washington, D. C. on Friday, November 21. The results of these examinations will be announced in a later issue of "Diseases of the Chest." *Report adopted.*

Conference of College Chapter Officials,
Nelson W. Strohm, M.D., Buffalo, New York, Chairman.

The Conference of College Chapter Officials reported in detail on the activities of the College chapters. A luncheon meeting of these officials was held at the Chelsea Hotel in Atlantic City on June 5, 1947. A great deal of discussion concerning the activities and the programs of the College chapters was presented at the meeting and it was planned that a similar meeting be held at the next annual meeting of the College in Chicago in June, 1948. *Report adopted.*

Committee on Certification,
J. Winthrop Peabody, M.D., Washington, D. C., Chairman.

The Committee on Certification reviewed in detail the steps that have been taken over a period of five years to obtain a recognized board on diseases of the chest, and reported that progress is being made towards accomplishing this objective. *Report adopted.*

Council on International Affairs,
Herman E. Hilleboe, M.D., Albany, New York, Chairman.

The officers of the Council on International Affairs met jointly with the officers of the Councils on Pan American, European and Pan Pacific Affairs and reviewed the progress that has been made by the College

in countries other than the United States of America. Plans are being developed for the expansion of College activities in the various countries. The following resolution was proposed:

> WHEREAS, The American College of Chest Physicians has members in 44 countries, and,
> WHEREAS, It is the aim of the American College of Chest Physicians to create a world organization of qualified chest specialists, and
> WHEREAS, The present councils dealing with international affairs of the College do not adequately cover all of the countries in the world, and
> WHEREAS, At the present time there are members of the College in South Africa, Egypt and Lebanon, and
> WHEREAS, These and other countries in this territory do not properly fit into the present arrangement of councils,
> THEREFORE, BE IT RESOLVED, That the Board of Regents of the College authorize the establishment of a Council on African and Near East Affairs and that the officers of this council be made members of the Council on International Affairs of the College.
> *Report and Resolution adopted.*

Research Council,
Jay Arthur Myers, M.D., Minneapolis, Minnesota, Chairman.

The Research Council reported the receipt of $17,940 in cash and pledges during the year 1947, and it was proposed that the campaign for research funds among the members of the College be intensified in order to reach the goal of $100,000 in the shortest period of time. A number of research projects were discussed and were referred to the Research Council for further consideration. *Report adopted.*

Council on Public Relations,
William C. Voorsanger, M.D., San Francisco, California, Chairman.

A five-point program was proposed by the Council on Public Relations. After considerable discussion this report was referred to the Executive Council of the College for further study.

Committee on College By-Laws,
Charles M. Hendricks, M.D., El Paso, Texas, Chairman.

The Committee on College By-Laws met on Friday evening, November 22, and discussed the suggested revisions in order to bring the present by-laws up to date. As a great deal of work remains to be done in connection with the activities of this committee, it was recommended that the committee report to the Board of Regents at the annual meeting of the College in Chicago in June, 1948.

Scientific Program Committee,
Richard H. Overholt, M.D., Brookline, Massachusetts, Chairman.

The Scientific Program Committee reported that preliminary plans for the program at the next annual meeting of the College are well under way and that the program this year will permit more time for the discussion of the papers presented. There will also be a number of interesting symposia on topics of current interest and the program will be international in scope. *Report adopted.*

Relationship of the American College of Chest Physicians with other Societies.

After considerable discussion of this subject, the Board of Regents

renewed its pledge to cooperate with all other societies concerned with the same objectives as the American College of Chest Physicians.

Dr. Andrew L. Banyai presented the following resolution regarding the Hyman I. Spector Memorial Collection:

> WHEREAS, The American College of Chest Physicians has been expanding its medical educational efforts throughout the nation and abroad and the prospects are that these endeavors are likely to attain increasingly greater momentum, and
> WHEREAS, Certain technical facilities are indispensable for the successful progress of such program, and
> WHEREAS, The office of the Executive Secretary serves as a center of an intensified postgraduate educational campaign in cooperation and in accordance with the plans of the Council on Postgraduate Medical Education, and
> WHEREAS, It seems desirable to acquire a collection of interesting lantern slides of particular teaching value which could be made available by lending to all Chapters of the College, to all recognized medical schools, and to all members of this organization without cost, and
> WHEREAS, It is appropriate to cherish the memory of the late Dr. Hyman I. Spector, former Regent of the College and former Chairman of the Examining Board of the College, whose name as a scientist has been generally recognized in medical circles, whose devotion to the College was a shining example of loyalty and solidarity and who spared no time, expense and effort in advancing the lofty aims of this organization in the service of medical science and humanity,
> THEREFORE, BE IT RESOLVED, By the Board of Regents of the American College of Chest Physicians that a collection of teaching lantern slides and other pertinent material be established as part of the College Library, under the auspices of the Council on Postgraduate Medical Education, to be known as the "Hyman I. Spector Memorial Collection" and that every effort be made to maintain and enlarge said Collection by the cooperation and generosity of all concerned.

There being no further business, the meeting was adjourned.

Meeting of Regents and Governors in Southern States

A meeting of the Regents and Governors of the College in the Southern States was held at the Sheraton Belvedere Hotel, Baltimore, Maryland, on Sunday, November 23, 1947, at the time of the meeting of the Southern Chapter of the College. Those present at the meeting were:

Regents

Paul A. Turner, M.D., Louisville, Kentucky,
Retiring President of the Southern Chapter, presiding
Dean B. Cole, M.D., Richmond, Virginia
Herbert L. Mantz, M.D., Kansas City, Missouri
Karl Schaffle, M.D., Asheville, North Carolina

Governors

Carl C. Aven, M.D., Atlanta, Georgia
Otto C. Brantigan, M.D., Baltimore, Maryland
R. Kyle Brown, M.D., Greenville, South Carolina
Edgar Davis, M.D., Washington, D. C.
M. Jay Flipse, M.D., Miami, Florida
E. C. Harper, M.D., Richmond, Virginia
R. O. Joplin, M.D., Louisville, Kentucky (Alt.)
George R. Maxwell, M.D., Morgantown, West Virginia
David H. Waterman, M.D., Knoxville, Tennessee

Guests

S. U. Marietta, M.D., Washington, D. C.
David Salkin, M.D., Hopemont, West Virginia
Walter E. Vest, M.D., Huntington, West Virginia

Dr. Turner called the meeting to order and introduced Major General S. U. Marietta, President of the College, who was guest speaker. General Marietta spoke about various activities of the College and a general discussion followed.

COLLEGE CHAPTER NEWS

ILLINOIS CHAPTER

The Illinois Chapter of the College sponsored a scientific meeting at the Congress Hotel, Chicago, on Friday evening, December 5. A dinner was held at the Congress Hotel at 6:30 p. m. and the following scientific program was presented at 8:00 p. m.:

The Industrial and Insurance Aspects of Chest Diseases

Industry: Harold A. Vonachen, M.D., Medical Director, Caterpillar Tractor Company, Peoria, Illinois.

Insurance: Roy W. Benton, M.D., Assistant Medical Director, Northwestern Mutual Life Insurance Company, Milwaukee, Wisconsin.

Chest Specialist: David F. Loewen, M.D., F.C.C.P., Medical Director, Macon County Tuberculosis Sanatorium, Decatur, Illinois.

The panel discussed cases, x-ray films, and questions presented by physicians in the audience. All physicians were invited to bring problem cases and x-ray films for discussion.

On Thursday, January 8, 1948, the Illinois Chapter of the College and the Chicago Tuberculosis Society sponsored a meeting at the Congress Hotel at which the following program was presented:

Symposium — Experience with Streptomycin in the Treatment of Tuberculosis:

"Tuberculous Meningitis and Miliary Tuberculosis,"
Abraham A. Levinson, M.D., Attending Pediatrician, Michael Reese Hospital, and Associate Professor of Pediatrics, Northwestern University School of Medicine.

"Tuberculosis of the Larynx and Bronchi,"
Albert H. Andrews, M.D., F.C.C.P., Associate Clinical Professor of Broncho-Esophagology, University of Illinois College of Medicine.

"Acute Pneumonic Tuberculosis,"
Charles K. Petter, M.D., F.C.C.P., Medical Director, Lake County Tuberculosis Sanitarium, Waukegan, Illinois.

"Surgical Treatment of Pulmonary Tuberculosis,"
S. Allen Mackler, M.D., Attending Thoracic Surgeon, Cook County Hospital.

"Tuberculosis of the Genito-Urinary System,"
Frederick Lloyd, M.D., Consultant in Urology, Veterans Administration Hospital, Hines, Illinois, and Assistant Professor in Urology, Northwestern University School of Medicine.

"Orthopedic Tuberculosis,"
Felix Gansey, M.D., Consultant in Orthopedics, Edward Miller, M.D., Resident in Orthopedics, and Bradley Carr, M.D., Resident in Orthopedics, Veterans Administration Hospital, Hines, Ill.

Discussion.

NEW YORK STATE CHAPTER

The New York State Chapter of the College will hold a meeting on January 29, 1948 at the Hotel New Yorker, New York City, at which time the following program will be presented:

Symposium on Extra Pulmonary Complications:

"Tuberculosis of the Meninges,"
Edith S. Lincoln, M.D., New York, N. Y.

"Tuberculosis of Serous Membranes,"
Harry Wessler, M.D., New York, N. Y.

"Tuberculosis of the Larynx, Trachea and Bronchi,"
Speaker to be announced.

"Tuberculosis of the Urinary Tract and the Use of Cali Moogrol Oil Derivative and Streptomycin,"
George Slotkin, M.D., Buffalo, N. Y.

"Tuberculosis of the Bones and Joints,"
Speaker to be announced.

"Tuberculosis of the Gastrointestinal Tract,"
Speaker to be announced.

Discussion to be opened by Oscar Auerbach, M.D., Veterans Hospital, Castle Point, New York.

Dinner: Speakers, Herman E. Hilleboe, M.D., F.C.C.P., New York; S. U. Marietta, M.D., F.C.C.P., Washington, D. C., President, American College of Chest Physicians.

Dr. George Foster Herben, Yonkers, New York, is Chairman of the Scientific Program Committee and serving with him on this committee are Dr. James S. Edlin, New York City, and Dr. Donald R. McKay, Buffalo, New York.

PACIFIC NORTHWEST DISTRICT CHAPTER

At the Annual Meeting of the Pacific Northwest District Chapter of the College, which was held in Seattle, Washington, on October 30 and 31, 1947, the following officers were elected for the coming year:

Grover C. Bellinger, M.D., Salem, Oregon, President
W. Elliott Harrison, M.D., Vancouver, B. C., Vice-President
Florence A. Brown, M.D., Portland, Oregon, Secy.-Treas. (re-elected)

The local chapter of the American Trudeau Society met jointly with the Pacific Northwest District Chapter of the College and the Northwest Trudeau Society was organized. Officers of the newly created Northwest Trudeau Society are:

Byron F. Francis, M.D., Seattle, Washington
William Hatfield, M.D., Vancouver, B. C., President-Elect
Cedric Northrop, M.D., Seattle, Washington, Secretary-Treasurer.

ROCKY MOUNTAIN CHAPTER

Lorenz W. Frank, M.D., F.C.C.P., Denver, Colorado, President of the Rocky Mountain Chapter of the College has announced the following committee appointments for the coming year:

Medical Education Committee:

Fred R. Harper, M.D., Denver, Colorado, Chairman
Albert Guggenheim, M.D., Denver, Colorado
Ralph G. Rigby, M.D., Salt Lake City, Utah
John A. Sevier, M.D., Colorado Springs, Colorado

Membership Committee:
John S. Bouslog, M.D., Denver, Colorado, Chairman
Aidan M. Mullett, M.D., Colorado Springs, Colorado
Carl H. Gellenthien, M.D., Valmora, New Mexico
B. Thomas McMahon, M.D., Denver, Colorado

Program Committee:
Harold M. Van Der Schouw, M.D., Wheat Ridge, Colo., Chairman
John B. Grow, M.D., Denver, Colorado
H. C. Goodson, M.D., Colorado Springs, Colorado
Laurence J. Bernard, M.D., Albuquerque, New Mexico

Publicity Committee:
Allan Hurst, M.D., Denver, Colorado, Chairman
Arthur B. Gjellum, M.D., Del Norte, Colorado
John G. Wolf, M.D., Pueblo, Colorado

General Arrangements Committee:
Arnold Minnig, M.D., Denver, Colorado, Chairman
Phineas J. Sparer, M.D., Denver, Colorado
Leroy Elrick, M.D., Denver, Colorado
John A. Cremer, M.D., Denver, Colorado.

SOUTHERN CHAPTER

An excellent scientific program was presented at the Fifth Annual Meeting of the Southern Chapter of the College held at the Sheraton Belvedere Hotel, Baltimore, Maryland, November 23-24, 1947, under the chairmanship of David H. Waterman, M.D., F.C.C.P., Knoxville, Tenn. The arrangements for the meeting were handled by Otto C. Brantigan, M.D., F.C.C.P., Baltimore, Maryland, and his able committee. Dr. Brantigan presided at the Banquet and introduced the guest of honor, Alfred A. Blalock, M.D., Baltimore, Maryland. Dr. Blalock presented a very interesting motion picture on "The Surgical Treatment of Coarctation of the Aorta." The following officers were elected for the Southern Chapter for the ensuing year:

Herbert L. Mantz, M.D., Kansas City, Missouri, President
Dean B. Cole, M.D., Richmond, Virginia, First Vice-President
David H. Waterman, M.D., Knoxville, Tenn., Second Vice-President
Hollis E. Johnson, M.D., Nashville, Tenn., Secy.-Treas. (re-elected)

The committee appointments will be announced in a later issue of "Diseases of the Chest."

WISCONSIN CHAPTER

The Wisconsin Chapter of the College sponsored a Symposium on Diseases of the Chest before the Winnebago County Medical Society on Thursday, December 4, 1947. The following papers were presented:

"Pulmonary Blastomycosis,"
David D. Feld, M.D., F.C.C.P., Milwaukee.

"Cystic Disease of the Lung,"
George H. Jurgens, M.D., F.C.C.P., Milwaukee.

"Atelectasis and Pulmonary Carcinoma,"
Mischa J. Lustok, M.D., F.C.C.P., Milwaukee.

ARGENTINE CHAPTER

The Annual Meeting of the Argentine Chapter of the American College of Chest Physicians, took place in Buenos Aires on December 6, 1947. Dr. Chevalier L. Jackson, Chairman of the Council on Pan American Affairs of the College, who was visiting in South America at that time, participated in the meeting. Other officials of the Chapter present at the meeting were:

Dr. Gumersindo Sayago, Cordoba, Regent
Dr. Raúl F. Vaccarezza, Buenos Aires, Governor
Dr. Agustin Caeiro, Cordoba, Vice-President
Dr. Alvaro E. Bence, Buenos Aires, Secy.-Treas.

A scientific session was held at 10:30 a. m. on the subject of "Utilidad de la Auroterapia en el Tratamiento de la Tuberculosis Pulmonar." The speakers at this session were:

Dr. Antonio Cetrangolo y Juan Carlos Rey
Dr. Justo Lopez (h) y Abraham Schottlender
Dr. Gumersindo Sayago y Juan B. Rocca.

Further details of this meeting of the Argentina Chapter of the College will be published in a later issue of the journal.

SOUTH BRAZILIAN CHAPTER ORGANIZED

On September 28, 1947, in the city of Sao Paulo, Brazil, a new College Chapter of the American College of Chest Physicians was inaugurated. This inauguration took place in collaboration with the 4a. Conferencia Regional de Tuberculosis, of the Sociedade Brasilieira de Tuberculose—Centre de Estudos do Dispensario Clemente Ferreira, held at Sao Paulo, Brazil on September 20-27, 1947.

The new College Chapter is to be known as the South Brazilian Chapter, and will be comprised of the following states: Rio Grande do Sul, Santa Catarina, Parana, and Sao Paulo. The Chapter will be supervised by Dr. Eduardo Etzel, Governor of the College for the southern Brazilian states, and becomes the thirty-first chapter of the College to be organized, and the third chapter to be organized for Brazil. The other two chapters in Brazil are (1) The Central Brazilian Chapter, comprising the states of Rio de Janeiro, D. F., Estado Rio de Janeiro, Espirito Santo, Minas Gerais, Gois Grasso, and Mato Grasso, and (2) The North Brazilian Chapter, comprising the states of Bahia, Sergipe, Alagoas, Pernambuco, Paraciba, Rio Grande do Norte, Ceara, Piaui, Maranhiao, Para, and Amazonas.

Dr. Reginaldo Fernandes, Rio de Janeiro, is the Governor for the Central Brazilian Chapter, and Dr. Jose Silveira, of Salvador, Bahia, is the Governor for the North Brazilian Chapter. Dr. Affonso MacDowell, Rio de Janeiro, is the Regent of the College for Brazil, and Dr. Affonso MacDowell Filho, is the Corresponding Secretary for the College Chapters in Brazil.

The meeting at Sao Paulo was attended by a large number of members of the College from the other Brazilian Chapters. Further information about this important and significant event in the activities of the College in South America, will be published in a later issue of the journal.

North Brazilian Chapter Holds Meeting

A reunion of the members of the North Brazilian Chapter of the American College of Chest Physicians, took place in Bahia, during the week of December 21-27, 1947, this meeting being in collaboration with the "Second Semana Anti-Tuberculosa" in Bahia. The meeting was attended by a large number of members of the North Brazilian Chapter of the College, headed by Dr. Jose Silveira, Governor of the North Brazilian Chapter. A special study of the problem of vaccination with BCG was presented at this meeting.

Dr. Chevalier L. Jackson Visits Brazil

Dr. Chevalier L. Jackson, Chairman of the Council on Pan American Affairs of the College, held a conference in Sao Paulo with Dr. Eduardo Etzel, and Dr. Etzel stated that the activities of the South Brazilian Chapter are progressing satisfactorily, and that a number of new applications for membership in the College will be received at the College offices from this territory in the near future.

In Rio de Janeiro, a reception was given in honor of Dr. Jackson, at the Yacht Club, by members of the Central Brazilian Chapter, and following the reception, a meeting of the members of the College was held, at which time Dr. Jackson delivered a message to them regarding the activities of the Council on Pan American Affairs of the College.

MEETING, CENTRAL BRAZILIAN CHAPTER

S. Sebastiao Hospital, Rio de Janeiro, November 20, 1947. Officers of the Central Brazilian Chapter, American College of Chest Physicians.

CENTRAL AMERICAN CHAPTER ORGANIZED

The Central American Chapter of the American College of Chest Physicians was organized in the city of San Salvador, El Salvador, C. A., on November 14, 1947. The meeting was held in the Sanatorio Nacional Anti-Tuberculosis. This new Chapter now becomes the thirty-second College Chapter, and adds an important milestone towards a complete organization of College Chapters in the Western Hemisphere. The session was attended by Dr. Amadeo Vicente Mastellari, Regent of the College for Central America, and by the following Governors of the College in Central America:

Dr. Gonzalez Bonilla, San Salvador
Dr. Enrique Coronado Iturbide, Guatemala
Dr. Rene Vargas Lopez, Nicaragua
Dr. Maximo Carrizo, representing the Governor from Panama.

A large number of College members in Central America also attended the meeting. Following the reading and approval of the College by-laws, the physicians named below were elected to office:

President: Dr. Rafael Leal H., Guatemala
Vice-President: Dr. Maximo Carrizo, Panama
Secy.-Treas. (Provisional): Dr. Fausto Aguilar, Guatemala
Directors: Dr. Arturo Blanco Solis, Costa Rica
Dr. Hector Valdez, Santa Ana, San Salvador.

The meeting was held jointly with the Asociacion Centro Americana de Tisiologia, and it was decided to send the delegates of that body a congratulatory message wishing them success in their endeavors. To celebrate the inauguration of the Central American Chapter of the College, a banquet was held on the evening of November 14th. It was voted that the next meeting of the Chapter should be held in Guatemala City, Guatemala.

THIRD ANNUAL MEETING OF THE PERUVIAN CHAPTER

The Third Annual Meeting of the Peruvian Chapter of the American College of Chest Physicians, was held at the Central Dispensario, Lima, Peru, on December 26, 27 and 29, 1947. The following scientific program was presented:

December 26th

"Criterio de Hospitalizacion en Tuberculosis,"
Prof. Dr. Ovidio Garcia Rosell.

"Indice de infeccion y morbilidad tuberculosa en postulantes a la Universidad Nacional Mayor de San Marcos,"
Prof. Dr. Juan A. Werner.

"Investigacion de la infeccion tuberculosa entre los enfermos mentales del Hospital 'Larco Herrera,'"
Dr. Luis Cano Gironda.

"Importancia del despistaje y del factor cultura en la lucha antituberculosa,"
Dr. Luis E. Hubner.

December 27th

"Sobre un caso de probable consinofilia tropical,"
Dr. Max. Espinoza Galarza.

"Granulia pulmonar tuberculosa en la infancia,"
Dr. Horacio Cachay Diaz.

"Asociacion de tuberculosis y absceso pulmonar,"
Dr. Leopoldo Molinari B.

"Tomografia en lesiones pulmonares apicales,"
Dr. Mario Pastor B.

"Absceso pulmonar,"
Dr. Pedro Zevallos A.

December 29th

"Calcificaciones pleurales,"
Dr. Ramon Vargas Machuca.

"Neumotorax bilateral,"
Dr. Juan Escudero Villar.

"Un caso de empiema cronico,"
Dr. Victor M. Tejada.

"Neumoserosa como tratamiento de las pleuresias,"
Dr. Roman del Castillo.

The names of the new officers and directors of the Chapter will be announced at a later date.

POSTGRADUATE COURSE TO BE GIVEN IN URUGUAY

The Fifth Curso de Perfeccionamiento Terico Practico—Problemas actuales de Clinica Tisiologica, will be held at the Instituto de Tisiologia "Prof. Juan B. Morelli" (Director: Prof. Dr. Fernando D. Gomez), Facultad de Medicina de Montevideo, on Marzo 1 al 13 de 1948. The following members of the American College of Chest Physicians will participate in this program: Prof. F. D. Gomez, Prof. P. Purriel, Prof. A. R. Gines (Paraguay), Dr. A. Victorica, Dr. D. A. Piaggio, Prof. J. Soto Blanco, and Dr. V. Armand Ugon.

Dr. Chevalier L. Jackson Visits Uruguay

During his trip through South America, Dr. Chevalier L. Jackson held a conference with Dr. Fernando D. Gomez, Montevideo, Governor of the College for Uruguay, and Dr. Gomez stated that the chest specialists in Uruguay are now preparing to organize a Chapter of the College in that country. Further details of the organization of this new College Chapter will be published in a later issue of the College journal.

NEWS NOTES

An excellent postgraduate refresher course in pulmonary tuberculosis was presented at the State Sanatorium, Tom Green County, Texas, the week of November 17-22, 1947. A number of physicians attended the entire course and visiting physicians attended the daily meetings from San Angelo and other points in West Texas. The next postgraduate course for Texas physicians will be offered by the State Sanatorium in the spring, probably in April, 1948. Physicians may make reservations for this course by writing the Superintendent, State Sanatorium, Sanatorium, Texas.

Announcement has been made of the Mount Sinai-Duarte National Medical Center, which is a consolidation of the Jewish Consumptive Relief Association, the Los Angeles Sanatorium, and the Mount Sinai Hospital and Clinic. The national headquarters are at 208 West Eighth Street, Los Angeles, California.

A new journal entitled "Revista Americana de Tisiologia" is now being published in Havana, Cuba. Dr. Juan J. Castillo, a Fellow of the American College of Chest Physicians, is Director and Founder. This new journal will be of interest to members of the College throughout the world.

Walter E. Vest, M.D., F.C.C.P., Huntington, West Virginia, has been re-appointed as president of the Public Health Council of the West Virginia State Medical Society. He has been a member of the Council since 1933, and has served as its president since 1937.

At a combined meeting of the Alumni of the Massachusetts Eye and Ear Infirmary and the New England Otolaryngological Society, which was held in Boston on November 19, Arthur Q. Penta, M.D., F.C.C.P., of Schenectady, New York, presented a paper on "The Role of Bronchoscopy in Clinical Medicine and Surgery."

George Evans, M.D., F.C.C.P., Clarksburg, West Virginia, was the guest speaker at the regular quarterly dinner meeting of the Central West Virginia Medical Society, September 25, 1947, in Buckhannon. The title of his paper was "Spontaneous Pneumothorax."

The following members of the College participated in the program of the American Medical Association presented during the Interim Session at Cleveland, Ohio, January 5-8, 1948:

Alvan L. Barach, M.D., F.C.C.P., New York, N. Y., led a discussion at the Conference on Respiratory Infections.

Alton Ochsner, M.D., F.C.C.P., New Orleans, Louisiana, led a discussion on the "Care of Posthospitalized Patient with Cancer."

Herman E. Hilleboe, M.D., F.C.C.P., Albany, New York, presented a paper entitled "BCG and Tuberculosis."

Leo G. Rigler, M.D., F.C.C.P., Minneapolis, Minnesota, presented a paper on "What Can Be Seen in a Roentgenogram of the Chest?"

AMERICAN COLLEGE OF PHYSICIANS RESEARCH FELLOWSHIPS IN MEDICINE, 1948 AWARDS

The Board of Regents of the American College of Physicians, on the nomination of the Committee on Fellowship and Awards, awarded six Research Fellowships in Medicine for the year beginning July, 1948, at their meeting in Philadelphia on November 22 and 23, 1947. The awards were made to the following physicians:

Charles G. Campbell, M.D., Vancouver, B. C.
Frank H. Gardner, M.D., San Bernardino, California
Samuel P. Martin, M.D., Durham, North Carolina
Peritz Scheinberg, M.D., Miami, Florida
Lutfu L. Uzman, M.D., Istanbul, Turkey
John M. Weller, M.D., Ann Arbor, Michigan.